Complex Food Webs: The Role of Parasites

Mcquaid, Finn

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Christopher Finn M^{c}Quaid

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Dedicated to Grandpa Jim, Granny Shirley, Grandpa Ian and Granny Margaret

Summary

Parasites are vital aspects of an ecosystem, and yet have only recently begun to be included in theoretical studies of food webs. There are numerous reasons for this neglect, but recent interest has arisen in remedying the situation.

Key to the addition of parasites are various structural features that ecological networks display, and the mechanisms behind them. We look at two of these features, nestedness and downward asymmetry, and describe mathematically the forces that create them. We discover from the basic reproductive ratio that population dynamics are insufficient to drive nested and anti-nested patterns in host-parasite networks, and instead we demonstrate the manner in which adaptive dynamics may be used to explain patterns through the coevolution of species interaction traits. We use the same technique in a mutualistic network in order to compare results. Following this, we use the basic reproductive ratio to demonstrate how the population dynamics of infectious systems promote the presence of trophic parasites in particular interaction motifs, and discuss the implications of the addition of parasites to the stability of ecological networks as a whole.

In summary, we demonstrate that the optimal use of resources by species in order to promote population growth results in two important structural patterns of host-parasite networks. Insights gained from this motivate us to investigate the importance of parasites on models of food web dynamics, and in particular, their stability.

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CHAPTER 1

Introduction

1.1 Food Webs

In ecological modelling, ecosystems describe the network of interactions between different species. The nature of these interactions depends upon the flow of energy or resources between species, as well as the effect that this has upon individuals of each species. A lot of work in this area has been focused on food webs, which are traditionally thought of as networks of predator-prey interactions. However, this neglects other important forms of interaction in ecological networks, such as parasitism, mutualism and competition. Some approaches have concentrated on these interaction types alone, or attempted to include them in ecological networks, but these have not always been successful. In this thesis we consider host-parasite interactions, and to a lesser extent mutualistic interactions, attempting to address certain issues that influence their presence in ecological systems.

Understanding an ecosystem as a whole requires the identification of such aspects as interaction structure and strength and the flow of energy, as well as keystone species (Byers, 2009). An understanding of all of the elements and connections in an ecosystem can then provide predictive power when considering the effects of environmental fluctuations due to climate change, the introduction of invasive species and habitat disruption, all currently such contentious issues (Ings et al., 2009; Wood, 2006). Ecological systems are

often depicted as food webs, networks in which individuals are grouped together according to (trophic) species, which are linked via consumer-resource interactions (Pimm, 1982) and the flow of energy or biomass (Lafferty et al., 2006b). In order to understand ecosystems, it is important to be able to model their structure and dynamics, which are intrinsically linked by the trophic interactions that represent these transfer rates (de Ruiter et al., 2005a).

May (1972) previously investigated the relationship between the complexity and number of connections in a food web and its stability, raising questions concerning the manner in which stable, large-scale food webs are able to exist in real life. However, as May himself made clear, this work included a range of assumptions that are unlikely to apply to real food webs, such as a lack of trophic structure and no links between interaction coefficients and complexity, which greatly oversimplify the networks involved (Nunney, 1980). It has also been shown that, for some food web models (specifically those with non-random complexity, a low predator-to-prey biomass ratio, strong levels of self-regulation or low assimilation efficiencies) the opposite assumptions are, in fact, true (De Angelis, 1975; Neutel et al., 2007; Solé and Bascompte, 2006). For such systems, an increase in connectance leads to a corresponding increase in stability (De Angelis, 1975). Therefore, although we see some regular patterns across food webs, such as short chain lengths, similar proportions of species at different trophic levels, and a rarity of cycles (Cohen et al., 1990), we are still left with many questions regarding ecological networks; how are their complex interactions stable, what determines the structure of these interactions, and how does this affect the dynamics of the individual species within the food web?

Food webs also represent a method for analysing community dynamics and function (de Ruiter et al., 2005a), in addition to the complexity and stability of the system (de Ruiter et al., 2005b; Neutel et al., 2007). Many factors affect food web dynamics and structure, including biotic and abiotic factors, and the availability and use of resources (de Ruiter et al., 2005a; Polis, 1994). However, each factor has differing importance under different conditions, and many factors are interdependent (Polis, 1994). Food web complexity spreads the effects of productivity and consumption of resources throughout trophic levels; indeed even assuming distinct trophic levels exist may be too great a simplification to make of ecosystems, where features such as omnivory are often more important than they are credited as being (Polis

and Strong, 1996).

1.1.1 Models

A plethora of well studied food webs demonstrate the complex sets of interactions under discussion here (see, for example, Cohen et al., 1990; Goldwasser and Roughgarden, 1993; Havens et al., 1996; Lafferty et al., 2006a; Martinez, 1991; Memmott et al., 2000; Niquil et al., 1999; Paviour-Smith, 1955; Polis, 1991; Schoenly and Cohen, 1991; Thompson et al., 2005; Varley, 1970; Warren, 1989; Winemiller, 1990). These include coral reefs (Arias-González et al., 1997) and estuaries (Hall and Raffaelli, 1991; Huxham et al., 1996), lochs (Morgan and McLusky, 1974) and streams (Schmid-Araya et al., 2002; Sukhdeo and Hernandez, 2004; Thompson and Townsend, 2003). The application of food webs to ecological systems all around us is clear.

Theoreticians faced with this range of empirical data have proposed numerous ways in which to model food webs (see Stouffer, 2010) which we shall now discuss. As with ecological modelling in general, these can fall into the categories of either a systems approach, concerning the flow of energy through trophic levels, or a population dynamics approach, concerning the details of species and their interactions. In food web modelling, these are represented by stochastic static models, recreating structural attributes of food webs, and evolutionary and dynamic models, controlling the formation and expansion of networks and relating the dynamics of systems to their stability (Lawton and Warren, 1988; Petchey et al., 2008).

Recent theoretical work on food webs has focused on static networks, although with an eye to amending this. Static models consider the structure of interactions in a food web, and are based on simple rules which use the number of trophic species and links in a network, as well as a small number of key network properties, such as connectance, diet discontinuity and the probability of forbidden links, to assign niche values to species. These niche values then determine the presence and direction of potential interactions between species, and a network structure is generated accordingly (Lafferty et al., 2008 and see Stouffer et al., 2005). The network structure is analysed by looking at a range of summary statistics. These include the number of species at different trophic levels, degrees of omnivory and cannibalism, the mean and variabilities of chain length and generality, as well as a number

of other features (Williams and Martinez, 2000). A stricter measure of the system as a whole is also suggested by Allesina et al. (2008); the likelihood that a model generates the observed data for a network structure. Stochastic models such as the cascade model (Cohen et al., 1990), the niche model (Williams and Martinez, 2000), the nested hierarchy model (Cattin et al., 2004), the inverted niche model (Warren et al., 2010) and the probabilistic niche model (Williams et al., 2010) have all improved understanding of static food networks, and the relationship between their complexity and stability.

Dynamical evolutionary models often assume undirected evolution, and use speciation in species assembly or evolution models to introduce new species to systems at equilibrium (Kondoh, 2003; Loeuille and Loreau, 2005). The Tangled Nature model focuses on individuals and the interactions between them, with a view to studying the coevolution of the entire system and any speciation that occurs (Christensen et al., 2002). Many dynamical models have also been based on Lotka-Volterra equations and their stability (Lawton and Warren, 1988), although aspects such as metabolic rates and resource allocation have also come into consideration (Brown et al., 2004). Moore et al. (2005) have modelled soil communities, including bacterial and fungal pathways, in order to determine the effects of shifts in the dynamic stability of an ecological system. Petchey et al. (2008) have used foraging behaviour and body size (linking energy content, handling time, attack rate and population density) to focus on individual-level mechanisms, and the network structure that ensues. In addition to this, assembly and evolution models, using population dynamics to attempt to create food webs from small, simple systems, have been proposed to account for and describe food webs (Drossel and McKane, 2005; Rossberg, 2005).

1.1.2 Caveats

The aforementioned static models are useful in generating a set of nodes and links onto which non-linear differential equations may be mapped (Warren et al., 2010), and hence population dynamics followed (Brose et al., 2006). They rely on large amounts of empirical evidence for motivation, and are very useful in describing network structures, but all rely heavily on only a very little input data for each separate system, and fail to take into account factors such as the strength that different interactions may have (Sukhdeo, 2010). More

importantly, the static structure of food webs has become less useful as focus has shifted towards the effects of environmental change, spatial ecology and issues of biodiversity (de Ruiter et al., 2005b). On the other hand, dynamical models are very difficult to parameterise, and are often highly unwieldy for large networks.

Food webs must be used with care, as they obscure information on features such as abundance, rates, spatial aspects and relationship types; although they are less tractable, dynamical models, which may take some these into account, are therefore often encouraged (Woodward et al., 2005). Static models have relied heavily on estimators such as body size in constructing a trophic hierarchy (Williams et al., 2010; Woodward et al., 2005), where as many as 90% of predator-prey interactions were seen to be between a larger predator and a smaller prey when measured across aquatic, terrestrial, coastal and marine systems (Cohen et al., 1993). Biomass ratio of predator to prey in interactions also appears to be an important factor in the stability of networks, enabling populations to persist (Brose et al., 2006). However, models are increasingly attempting to use energy flow and metabolic rates to account for food web structure and dynamics, as opposed to the consideration of trophic levels (Allesina et al., 2008; Brown et al., 2004; Sukhdeo, 2010; Zhang and Guo, 2010). Both dynamical and structural aspects of a network have important effects on the stability of the network, particularly when taken together (Cattin et al., 2004), and both approaches have been used separately in the past in order to describe and explain patterns in food webs, with a fair amount of success. Yet each approach has its drawbacks, as noted above (see Lawton and Warren, 1988).

In particular, many of the previous models of ecological networks and food webs have neglected interactions of types other than predator-prey, such as mutualistic, parasitic and competitive interactions. Here we model host-parasite and mutualistic networks in a way that links dynamics (both population and evolutionary) to network structure. The focus of the biological world has become increasingly centred on adapting to dynamical food webs; the structure of a network arises as a result of the evolution of its dynamics, rather than *vice versa* (Warren et al., 2010; Drossel and McKane, 2005). Non-equilibrium dynamics are key to understanding the complex interactions of food webs (Dobson et al., 2009), as static models cannot explain structure, only predict it (Drossel and McKane, 2005). Food webs are instead

an emergent structure of individual-level mechanisms (Petchey et al., 2008). In general, it is felt that the interactions of individuals and their evolution ultimately gives rise to the complex structures that are our way of visualising ecosystems (Neutel et al., 2007).

1.2 Parasites in Food Webs

The role of parasites in ecological network models has largely been neglected in the past. Parasites represent a large amount of biomass and species diversity in ecological systems, and impact the relative abundance of free-living species (Kuris et al., 2008; Lafferty et al., 2006b; Poulin, 2010). Even current estimates of parasite presence in ecological systems may be too low, with many parasite species regularly escaping detection (Poulin, 1998a). Parasites affect the growth, reproduction and mortality of host species (Lafferty et al., 2006b), as well as reducing the transfer of energy from prey to predators (Brose et al., 2006). Some parasites may even be important in food webs as a prey source, such as the zoospores of chytrids, which provide food for zooplankton (Kagami et al., 2007). All of the above has far-reaching consequences, and the presence of parasites could even be destabilising for entire ecosystems (Brose et al., 2006), where they are also important in terms of species extinctions (De Castro and Bolker, 2005a,b). From a human perspective, the position of parasites in a food web is important as they are significant in diseases of humans, livestock, fishstock and agriculture (Marcogliese, 2002). They are indicators of diet and migration (Marcogliese and Cone, 1997), and indeed of habitat vulnerability, which may become increasingly important in revealing ecological restoration, as can be seen in trematodes in snails (Lafferty et al., 2008).

Looking at the inclusion of parasite species as a whole into food webs is of great importance, as it gives a more robust view of entire ecosystems, rather than just looking at unrealistic, isolated interactions (Poulin, 2010). Indeed, parasitism is the most common form of consumer-resource interaction (Lafferty et al., 2006b). In recent years there have been many calls to include parasites in food webs (Byers, 2009; Dobson et al., 2009; Lafferty et al., 2006b; Leaper and Huxham, 2002; Mouillot et al., 2008; Marcogliese and Cone, 1997). Although this has been investigated to some extent, especially with parasitoids (Lafferty et al., 2006b; van Veen et al., 2008), much

still remains to be explained (Lafferty et al., 2008). We must also be wary of using insights gained from macroparasitic systems, as their mechanisms are often different, and their effects on hosts may be density dependent, unlike pathogens (Lafferty et al., 2008). Parasites alter straightforward static network properties, such as species richness, link number and chain length (Lafferty et al., 2008; Thompson et al., 2005) as well as more important factors such as the connectance and nestedness (Lafferty et al., 2006b), which have been used to comment on the structure and stability of food webs in the past. Parasites may even affect community structure, trophic relationships and energy flow in entire ecosystems (Britton, 2013; Holt and Pickering, 1985; Lafferty et al., 2008; Marcogliese and Cone, 1997; Poulin, 2010; Thompson et al., 2005), making them highly significant.

It is a fundamental goal of ecology to understand complexity in ecological systems; how it can persist and the effects on function that it has (Ings et al., 2009). The complex interactions between stability, connectivity, species diversity and interaction strength are all thrown into disarray by the addition of parasite species (Lafferty et al., 2006b). Despite the fact that complexity in random-pattern models leads to instability (Neutel et al., 2007), it is thought that food webs may rely on complexity and diversity to provide stability, so the impact of parasites here is potentially vital (Wood, 2006). The question remains as to whether they affect the energy flow and stability of these systems (Byers, 2009), and there is a need to quantify this in order to place them correctly in food webs (Sukhdeo, 2010). This relates to interaction strengths, and the resilience of the network to environmental perturbations (Poulin, 2010). Theoretical models have shown that the stability of a network is highly influenced by the presence of many weak interactions, such as those represented by host-parasite links when compared to stronger predator-prey links, and variation in interaction strengths among network links (Poulin, 2010). Specialist species have stronger interactions than generalists, while generalists obviously have more (Montoya et al., 2006). In addition, a variety in interaction types (specifically antagonistic and mutualistic interactions) increases the stability of a system (Mougi and Kondoh, 2012, although see Suweis et al., 2013), and parasites shared between hosts may even be a strongly stabilizing factor (Lafferty et al., 2008). A low predator-prey biomass ratio also promotes stability as the complexity of a system increases, which will be affected by the presence of parasites (Neutel et al., 2007).

1.2.1 Problems

Although it appears that there may well be universal laws acting on host-parasite interaction networks (Poulin, 2007a), fitting parasites into food webs has its difficulties. The fundamental assumptions of the cascade model (Cohen et al., 1990) and models based on it are violated by the presence of parasites (Lafferty et al., 2008). Many of the static models discussed above are founded on a topological ordering based on body size, which is, of course, reversed in the case of parasites (Leaper and Huxham, 2002; Rossberg, 2005). Other methods of trophic ordering, such as the ratio of heavy nitrogen-15 to light nitrogen-14, are also less applicable to parasites, as they are very much dependent on both the parasite's host species and which part of the host organism it infects (Lafferty et al., 2008). In addition, parasites can have complex life-cycles, which involve multiple host species (Huxham et al., 1995). Grouping these life stages together makes parasites appear to be generalists, when they could be specialists in each life stage; separating life stages out requires complicated energy flow between the stages, which is difficult in terms of traditional food web tools, such as linear algebra and graph theory (Brose et al., 2006). Although transmission routes of parasites often follow the trophic pathways that link hosts (Morozov and Adamson, 2011; Poulin, 2010; Thompson et al., 2005), parasites cannot simply be pasted onto existent food webs (Sukhdeo, 2010). Parasitism often leaves hosts more vulnerable (Minchella, 1985), but parasites themselves are also vulnerable to secondary extinctions if their host species is threatened (Lafferty et al., 2008). Parasites, therefore, have a marked effect on their hosts, and *vice versa* (Morozov and Adamson, 2011).

1.2.2 Previous work

Examples exist of the inclusion of parasites, but few from a dynamical modelling perspective. Warren et al. (2010) investigated a salt marsh system, and in an effort to include parasites used the niche model to construct a static network by reversing the trophic reliance on body size as an ordering for parasitic interactions. Memmott et al. (2000) included parasites in a manner similar to the cascade model by creating separate subwebs for different interaction types; predatory or parasitic. Huxham et al. (1995) included parasites in the Ythan estuary and Loch Leven food webs, again from a static per-

spective, and Hernandez and Sukhdeo (2008) included helminth parasites in a stream food web. Cumming and Guégan (2006) made a pan-African study of pathogens and their vectors, ticks, relating this to species richness and the abiotic environment, but focused on the host-vector-pathogen system and not the entire food web. None of these approaches seem to capture the essence of the interactions here, or to explain the dynamics of the species involved.

An increase in practical and statistical studies including parasites has left a deficit of mathematical models to account for observable patterns (Chen et al., 2008). Many models focus on single-species systems, or static networks, due to the intractability of larger, dynamical networks. Some look at pathogens with multiple hosts (Dobson, 2004; Gandon, 2004; Osnas and Dobson, 2011), or hosts with multiple parasites (see Poitrineau et al., 2003; Pugliese, 2002), but few investigate full systems with both multiple hosts and parasites (although see Frank, 2000; Garnick, 1992). Even those which include multiple species look solely at host-parasite interactions, and not at the network as a whole (Poulin, 2010).

1.3 Thesis Outline

The challenge remains, therefore, to introduce parasites to dynamical food webs. With this in mind, we will focus on two particular structural patterns that can be observed in the occurrence of parasites in food webs, as well as investigating their effect on food web stability. One of the structural aspects focuses on the relationship between sets of parasite species infecting different host species, and the second on the effects of the food web structure on parasitic composition. Chapters 3, 4, 5 and 6 exist as self-contained, published articles, and hence contain some overlap in their introduction and modelling sections.

The first pattern that we investigate is called nestedness. We outline what this means for a host-parasite system, some of the conflicting evidence for patterns of nestedness in both host-parasite systems and beyond, and propose a potential evolutionary explanation.

Nestedness, in a general system, occurs when those species occurring in a species-poor assemblage form a subset of those assemblages with a higher species richness (Poulin and Guégan, 2000). In other words, for a host-parasite system, the parasites of hosts attacked by few species are also found

parasitizing hosts with many parasitic species. In a similar, although slightly weaker, case, generalist parasites are often found in hosts with both high and low parasite species diversity, while specialist parasites are found mainly in hosts with a rich diversity of parasites (Poulin, 1997; Vázquez et al., 2005). This association, between the specificity of parasites and the species-richness of the hosts that they infect, is known as specialization asymmetry (Vázquez et al., 2005).

These patterns of nestedness and specialization asymmetry can be seen from parasites in fish species (Poulin, 1997) to fleas and their hosts (Vázquez et al., 2005), although there is some debate on the extent to which this is evident, and counterexamples exist (Poulin, 1997, 2007a; Valtonen et al., 2001). In fact, there appears to be evidence for both significant amounts of nestedness and anti-nestedness in host-parasite systems (Graham et al., 2009; Joppa et al., 2010; Poulin, 2007a; Poulin and Guégan, 2000). Unfortunately, definitions for anti-nestedness vary depending on the metrics used for measurement (Almeida-Neto et al., 2007). For example, different metrics consider anti-nestedness to refer to random assemblages, compartmentalised networks, those with a perfect gradient, or species-absence matrices. Nevertheless, we ask what drives this association between the host-specificity of parasites and the parasite species-richness of the hosts that they infect.

Nestedness is also evident in many other ecological networks (Ings et al., 2009; Kondoh et al., 2010; Warren et al., 2010), and is a prevalent feature of mutualistic networks in particular (Bascompte et al., 2003; Vázquez and Aizen, 2003, 2004). This is interesting, given the complete reversal in interaction types involved when compared to host-parasite networks. There is mixed evidence for whether the addition of parasites to these webs should increase or decrease their relative nestedness (Hernandez and Sukhdeo, 2008; Lafferty et al., 2006b), as antagonistic networks are generally expected to be compartmentalized (Bascompte, 2010; Thébault and Fontaine, 2008; Thompson, 2005; but see Kondoh et al., 2010; in addition see Flores et al., 2011 for the effects of scale-dependence). In order to fit parasites into food web models, it is important to understand the forces behind such structural traits as nestedness, as nested networks could be used as building blocks for complex food webs (Kondoh et al., 2010).

We wish, therefore, to investigate the impact of the addition of parasite species on the behaviour of a food web. In order to do so, we begin in Chap-

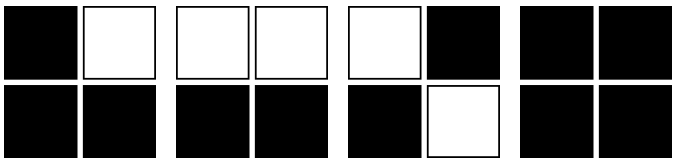


Figure 1-1: Possible interaction matrices for a 2-host (y-axes) 2-parasite (x-axes) network. A black square represents an interaction between two species and a white square no interaction. Plots demonstrate specialization asymmetry with (a) a generalist and a specialist parasite (also displaying nestedness) and (b) two specialist parasites in the same host, and specialization symmetry with (c) two specialist parasites in opposite hosts and (d) two generalist parasites.

ter 2 by focusing on a system containing multiple host and multiple parasite species, with no apparent structure of host interactions. The simplest such case that we can find contains two host and two parasite species; this allows for specialist and generalist species while still remaining relatively uncomplicated. This system is also the simplest that can display specialization asymmetry, and indeed nestedness (see figure 1-1). We attempt to calculate the basic reproductive numbers for parasites in systems showing nestedness or specialization asymmetry, and compare them to parasites in other two-host, two-parasite systems.

We do not find sufficient evidence from the basic reproductive number to suggest that a nested host-parasite system is at any particular advantage, so in Chapter 3 (McQuaid and Britton, 2013a) we propose an exploratory coevolutionary model of the dynamics of the system. Nestedness can be linked to the host range of parasites, which has been related to foraging theory (Byers, 2009), motivating us to use a trade-off in resources in a similar manner. Our model proposes that host-parasite network structure is an emergent property of species trade-offs in the manner with which they interact with other species, and the evolution of these trade-offs over time. The model is analysed in detail, and then expanded in Chapter 4 (McQuaid and Britton, 2013b) to a higher dimension. We observe that the presence of parasite species affects host immune systems, and hence influences infection by other species. In Chapter 5 (McQuaid and Britton, 2013c), the core ideas in this evolutionary investigation are transferred to a mutualistic system for comparison. We conclude that a trade-off in resource use could indeed explain nestedness in both host-parasite and mutualistic networks.

Ideally, in the theory of food webs, an integrated model based on individual antagonistic and mutualistic species interactions should be used as a descriptor. These would be entire networks, which react over evolutionary time, instead of representing simply a snapshot, and which could be used for prediction based on mechanistic principles (Ings et al., 2009). An extension to our work could demonstrate a potential line of research for this in the future, dynamically modelling a general ecosystem with no prior assumptions on interaction types in the same manner as described in Chapters 3-5.

Following this, in Chapter 6 we address a second observed structural pattern of parasites in food webs. Chen et al. (2008) have shown that host position in a network (with respect to its predators and prey) is an important determinant when considering its parasite diversity; the wider the diet range of a host, or the greater the proximity of the host to prey species, the higher the diversity of parasites infecting it. Hosts which are vulnerable to predators also tend to be important in parasite transmission. In particular, predator-prey interactions where the predator has many prey species, but each prey species has few predators, have been shown to be particularly rich in trophically-transmitted parasite species (Rossiter and Sukhdeo, 2011). In Chapter 6 (McQuaid and Britton, 2013d) we explore the presence of this pattern as a consequence of an increase in the basic reproductive ratio.

Finally, in Chapter 7, in a reversal of the effects of stability on parasite persistence and species richness that we investigate in Chapter 6, we look at the effects of parasites on the stability of ecological networks. Mougi and Kondoh (2012) have investigated the effects of mixing mutualistic and antagonistic interaction types in an ecological network, and suggest that this increases the local asymptotic stability of these systems (although see Suweis et al., 2013). We extend the concept here to include parasitic interactions, and look at the effect that this has on persistence in systems. We conclude that the addition of parasitic interactions has a small but noticeable effect on the persistence of free-living species in an ecological network, although this is dependent on the type of interactions present in the network.

Combining the results of our studies, we see that the presence of parasites in food webs is affected by both host species and other parasite species, as well as ecological and evolutionary dynamics. We have used foraging theory, the basic reproductive ratio and persistence to investigate and explain structural aspects of parasite species' richness and its effect on stability, and use this

to describe where we would expect to see congregation of parasitic species. These features are vital in including parasites in food webs, as they provide mechanistic principles for the location, function and effects of the parasites. We finish in Chapter 8 by drawing conclusions on some important features of host-parasite networks, and the manner in which they fit into food webs.

CHAPTER 2

Nestedness and the Basic Reproductive Number

One of the most-used indices in host-parasite systems is the basic reproductive number, R_0 (Diekmann et al., 2010). This is defined as "the expected number of secondary cases produced by a typical infected individual during its entire period of infectiousness in a completely susceptible population" (Diekmann et al., 1990). Thus, the basic reproductive number may be used to measure the "success" of an infectious parasite, and can determine whether or not the infection will spread in a susceptible population. Previously, this has been calculated for systems with multiple hosts (Dobson, 2004; Holt et al., 2003) or multiple parasites (Poitrineau et al., 2003; van Baalen and Sabelis, 1995), but the workings of a system with both multiple hosts and multiple parasites remain relatively obscure (although see Osnas and Dobson, 2011; Zhang et al., 2007). In order to fully understand the place of parasites in food webs, it is important for us to take into consideration the impact on R_0 of high species dimension. Here we look at the relative values of R_0 for different structures of host-parasite systems, in an attempt to explain the prevalence of the structural pattern nestedness; if a nested system results in a higher basic reproductive number for parasites than an anti-nested system, then we might expect nestedness to abound. However, we discover that there is almost no evidence for an increase in parasite success in a nested system, and conclude that forces other than a simple increase in basic reproductive number must instead promote nested patterns.

2.1 The Model

Our model is based on a system with two host species and two parasite species. The parasites may be transmitted both within and between host species, and in a simplification, the susceptibility of an individual to a parasite species is assumed to be unaffected by the presence or absence of the other parasite species. A susceptible-infectious (SI) model is assumed for $i, j \in \mathbb{N}_2$ as indices for the host and parasite species respectively, leading to the following system of differential equations:

$$\begin{aligned}
\frac{dS_i}{dt} =& b_i - \sum_{j\in\mathbb{N}_2}\sum_{k\in\mathbb{N}_2} \beta_j^{ik} I_{kj} S_i - \sum_{k\in\mathbb{N}_2} \frac{\beta_1^{ik}+\beta_2^{ik}}{2} C_k S_i - d_i S_i, \\
\frac{dI_{ij}}{dt} =& \sum_{k\in\mathbb{N}_2} \beta_j^{ik} I_{kj} S_i - (d_i+\gamma_{ij}) I_{ij} - \sum_{k\in\mathbb{N}_2} \beta_{j-1}^{ik} C_k I_{ij} - \sum_{k\in\mathbb{N}_2} \beta_{j-1}^{ik} I_{kj-1} I_{ij}, \\
\frac{dC_i}{dt} =& \sum_{k\in\mathbb{N}_2} \frac{\beta_1^{ik}+\beta_2^{ik}}{2} C_k S_i + \sum_{j\in\mathbb{N}_2}\sum_{k\in\mathbb{N}_2} \beta_j^{ik} C_k I_{ij} + \sum_{j\in\mathbb{N}_2}\sum_{k\in\mathbb{N}_2} \beta_j^{ik} I_{kj} I_{ij-1} \\
& - (d_i+\psi_i) C_i,
\end{aligned} \tag{2.1.1}$$

Here, S_i represents susceptibles of host type i, I_{ij} indicates host type i infected with parasite type j, and C_i represents host type i infected with both parasites. Transmission parameter β_j^{ik} represents the acquisition of parasite j by host type i from host type k, and we make the assumption that coinfection occurs at the average rate of infection for each parasite species. Births occur into the susceptible categories at rates b_i for host i, and deaths occur naturally at a per capita rate d_i, with an increased rate due to the presence of parasites. The increase in mortality due to parasites is γ_{ij} for host i due to parasite j, and is ψ_i for host i due to coinfection.

Susceptible individuals are born, become infected or coinfected, and die. Infected individuals are infected, die due to natural causes or infection-induced mortality, or are coinfected with a second parasite species after contact with either a coinfected individual or one infected with the second parasite species only. Individuals become coinfected due to either contact with a coinfected individual, or two subsequent infections by opposite parasite species. They then die of either natural or disease-related causes.

2.2 Analysis

We begin by investigating a simplified system, defining it such that the first parasite species may infect both host species, and is therefore considered a generalist parasite, while the second parasite species may only infect the second host, and hence, by comparison, is a specialist parasite (see figure 2-1). This implies that

$$\beta_2^{11} = \beta_2^{12} = \beta_2^{21} = 0. \tag{2.2.2}$$

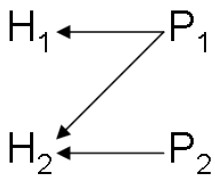


Figure 2-1: A two-host, two-parasite model where arrows represent interactions between host and parasite species. Host species H_1 has poor parasite species richness, while host species H_2 has high parasite species richness. Likewise, parasite species P_1 is a generalist, while parasite species P_2 is a specialist.

The first host species therefore has low parasite species richness, while the second has a high level of parasite species richness. This is the simplest system in which we can see the parasitic composition of one host species as a proper subset of the other (resulting in the closest approximation to nestedness that such a small system can have), so we begin by investigating the basic reproductive numbers for both parasites in this system.

We do not divide our basic reproductive number so that each parasite has a different ratio for different hosts, as Garnick (1992) has in a similar model lacking coinfection. We focus instead on each parasite and its ability to establish itself in the community as a whole when rare. From our system, the basic reproductive number for each parasite, R_0, is determined by calculating the dominant eigenvalue of the next-generation matrix (Diekmann and Heesterbeek, 2000). This is a matrix that relates the number of infected individuals in different compartments between generations (Diekmann et al., 2010).

For this, we focus on the infected subsystem, considering the population dynamics of infected individuals of different classes only, linearized about the infection-free steady state. The linearized system of ordinary differential equations can then be described by the next-generation matrix (NGM). The ijth element of the NGM consists of the rate of infection of infectious type i by type j, multiplied by the duration of time for which the infected individual i remains in its current state (Dobson, 2004). The duration is influenced

by the death rate, as well as the possibility of a singly infected individual being further infected with a second parasite species, and hence becoming coinfected. For a detailed description of the construction and application of NGMs to the basic reproductive number, see Diekmann et al. (2010), and, originally, Diekmann et al. (1990).

NGMs are constructed below, and simplifications introduced in order to compare results to those for well-known systems. While specific R_0 equations for the full system are not included, due to their size and complexity, the conditions under which a parasite species may exist are drawn from these equations and discussed in section 2.3.

2.2.1 Basic reproductive number of first parasite

It is presumed that the first parasite has entered the system at a point where the second parasite is at a non-trivial equilibrium in that population, and that the first parasite is at such low levels as to make it negligible. The non-trivial equilibrium for a system containing the second parasite only occurs at:

$$S_1^* = \frac{b_1}{d_1}, \qquad S_2^* = \frac{d_2 + \gamma_{22}}{\beta_2^{22}} \qquad \text{and} \qquad I_{22}^* = \frac{b_2}{d_2 + \gamma_{22}} - \frac{d_2}{\beta_2^{22}}.$$

The NGM for the first parasite is given by:

$$K_1 = \begin{pmatrix} \frac{\beta_1^{11} S_1^*}{(d_1+\gamma_{11})} & \frac{\beta_1^{12} S_1^*}{(d_2+\gamma_{21}+\beta_2^{22} I_{22}^*)} & \frac{\beta_1^{12} S_1^*}{(d_2+\psi_2)} \\ \frac{\beta_1^{21} S_2^*}{(d_1+\gamma_{11})} & \frac{\beta_1^{22} S_2^*}{(d_2+\gamma_{21}+\beta_2^{22} I_{22}^*)} & \frac{\beta_1^{22} S_2^*}{(d_2+\psi_2)} \\ \frac{\beta_1^{21} I_{22}^*}{(d_1+\gamma_{11})} & \frac{(\beta_2^{22}+\beta_1^{22}) I_{22}^*}{(d_2+\gamma_{21}+\beta_2^{22} I_{22}^*)} & \frac{\beta_1^{22} I_{22}^*}{(d_2+\psi_2)} \end{pmatrix}. \tag{2.2.3}$$

R_0 may then be determined by calculating the dominant eigenvalue of K_1. We show some simple cases for R_0, where the number of species has been reduced. These can be easily compared both to known problems and systems constructed specifically for these conditions.

One host, one parasite:

If it is presumed that there is no second host, then a simple one-host one-parasite system results, as the second parasite may not infect the first host. Note that this requires a birth rate of zero for the host. The basic reproductive number for this may be readily checked, and does indeed confirm the result

obtained if it is presumed that

$$S_2^* = I_{21}^* = I_{22}^* = C_2^* = 0,$$

which yields

$$\lambda = \frac{S_1^* \beta_1^{11}}{(d_1 + \gamma_{11})}. \tag{2.2.4}$$

Two hosts, one parasite:

If it is presumed that the second parasite is at a trivial equilibrium, then assuming

$$I_{22}^* = C_2^* = 0$$

simplifies the expression for K_1 in equation 2.2.3, and the dominant eigenvalue is given by

$$\begin{aligned} \lambda &= \frac{1}{2}\left(\frac{S_1^* \beta_1^{11}}{(d_1 + \gamma_{11})} + \frac{S_2^* \beta_1^{22}}{(d_2 + \gamma_{21})} \right) \\ &+ \frac{1}{2}\sqrt{\left(\frac{S_1^* \beta_1^{11}}{(d_1 + \gamma_{11})} - \frac{S_2^* \beta_1^{22}}{(d_2 + \gamma_{21})} \right)^2 + \frac{4 S_1^* \beta_1^{12} S_2^* \beta_1^{21}}{(d_2 + \gamma_{21})(d_1 + \gamma_{11})}}. \end{aligned} \tag{2.2.5}$$

Alternatively, the NGM for a system with two host and one parasite species yields a similar result, and indeed equation 2.2.5 may be adjusted to render it identical to the result obtained by Dobson (2004).

One hosts, two parasite:

In a final comparison, if it is presumed that the first host is at a trivial equilibrium (again requiring a birth rate of zero), then

$$S_1^* = I_{11}^* = 0.$$

This corresponds to the situation where one host is infected by two parasites. The dominant eigenvalue of equation 2.2.3 is found to be:

$$\lambda = \frac{1}{2}\left(\frac{I_{22}^{*}\beta_1^{22}}{(d_2+\psi_2)} + \frac{S_2^{*}\beta_1^{22}}{(d_2+\gamma_{21}+\beta_2^{22}I_{22}^{*})}\right) + \frac{1}{2}\sqrt{\left(\frac{I_{22}^{*}\beta_1^{22}}{(d_2+\psi_2)} + \frac{S_2^{*}\beta_1^{22}}{(d_2+\gamma_{21}+\beta_2^{22}I_{22}^{*})}\right)^2 + \frac{4S_2^{*}\beta_1^{22}I_{22}^{*}\beta_2^{22}}{(d_2+\gamma_{21}+\beta_2^{22}I_{22}^{*})(d_2+\psi_2)}}, \tag{2.2.6}$$

which can be confirmed by considering the dominant eigenvalue of the 2x2 NGM for a system with one host and two parasites, given by

$$K_{12} = \begin{pmatrix} \frac{\beta_1^{22}S_2^{*}}{(d_2+\gamma_{21}+\beta_2^{22}I_{22}^{*})} & \frac{\beta_1^{22}S_2^{*}}{(d_2+\psi_2)} \\ \frac{(\beta_2^{22}+\beta_1^{22})I_{22}^{*}}{(d_2+\gamma_{21}+\beta_2^{22}I_{22}^{*})} & \frac{\beta_1^{22}I_{22}^{*}}{(d_2+\psi_2)} \end{pmatrix}.$$

One final way to confirm this is to compare it to the result obtained for the basic reproductive number of the second parasite under similar conditions. If there is only one host, then the two parasites will act in an identical manner, and hence should yield an R_0 of a similar form. This will be discussed later, but we note here that it is indeed the case.

2.2.2 Basic reproductive number of second parasite

The NGM for the second parasite is now obtained and compared for different submodels. At a steady state, with $S_1^{\dagger}$, $S_2^{\dagger}$, $I_{11}^{\dagger}$ and $I_{21}^{\dagger}$ denoting the equilibrium values for S_1, S_2, I_{11} and I_{21} respectively, the NGM is given by

$$K_2 = \begin{pmatrix} \frac{\beta_2^{22}S_2^{\dagger}}{(d_2+\gamma_{22}+\beta_1^{22}I_{21}^{\dagger}+\beta_1^{21}I_{11}^{\dagger})} & \frac{\beta_2^{22}S_2^{\dagger}}{(d_2+\psi_2)} \\ \frac{(\beta_2^{22}+\beta_1^{22})I_{21}^{\dagger}+\beta_1^{21}I_{11}^{\dagger}}{(d_2+\gamma_{22}+\beta_1^{22}I_{21}^{\dagger}+\beta_1^{21}I_{11}^{\dagger})} & \frac{\beta_2^{22}I_{21}^{\dagger}}{(d_2+\psi_2)} \end{pmatrix}. \tag{2.2.7}$$

One host, two parasites:

In this case, the parasite has no host species, and therefore cannot establish itself. Setting

$$S_2^{\dagger} = I_{21}^{\dagger} = I_{22}^{\dagger} = 0,$$

the eigenvalues do indeed all equate to zero as expected.

Two hosts, one parasite:

For the case where the first parasite is at a trivial equilibrium, the solution should simply be the same as that for a one-host, one-parasite system, as the parasite may not infect the first host. Letting

$$I_{11}^{\dagger} = I_{21}^{\dagger} = C_2^{\dagger} = 0,$$

it is found that

$$\lambda = \frac{\beta_2^{22} S_2^{\dagger}}{(d_2 + \gamma_{22})},$$

as is expected for such a system; this can also be compared to equation 2.2.4 above.

One host, two parasites:

Finally, when the first host is absent, we should find identical results to the same case for the basic reproductive number of the first parasite (equation 2.2.6). Setting

$$S_1^{\dagger} = I_{11}^{\dagger} = 0, \tag{2.2.8}$$

it can be seen that

$$\lambda = \frac{1}{2}\left(\frac{\beta_2^{22} S_2^{\dagger}}{(d_2 + \gamma_{22} + \beta_1^{22} I_{21}^{\dagger})} + \frac{\beta_2^{22} I_{21}^{\dagger}}{(d_2 + \psi_2)}\right) + \frac{1}{2}\sqrt{\left(\frac{\beta_2^{22} S_2^{\dagger}}{(d_2 + \gamma_{22} + \beta_1^{22} I_{21}^{\dagger})} + \frac{\beta_2^{22} I_{21}^{\dagger}}{(d_2 + \psi_2)}\right)^2 + \frac{4\beta_2^{22} S_2^{\dagger} \beta_1^{22} I_{21}^{\dagger}}{(d_2 + \gamma_{22} + \beta_1^{22} I_{21}^{\dagger})(d_2 + \psi_2)}}. \tag{2.2.9}$$

This is indeed of the same form as for the first parasite, and confirms that the NGMs for the parasites concur. This can also be checked by constructing an NGM for the situation where there is only one host. This is exactly the same NGM as equation 2.2.7 when applying condition 2.2.8, and hence yields the same result.

It is also worth noting that, due to the assumption of resource specialization, we presume that the specialist parasite must out-compete the generalist in the same host, i.e. the second host (Garnick, 1992). Hence, we require that basic reproductive numbers satisfy $\lambda_{2.2.6} < \lambda_{2.2.9}$.

All of these simplifications serve to emphasize the importance of considering a full system when calculating the NGM, and we now turn the NGM to the problem at hand; identifying and analysing the R_0 of a full, multi-host, multi-parasite system.

2.3 Results

As there is no direct competition between the parasites, if R_0 for each parasite is greater than 1 then each will always be able to grow in a susceptible population. Unlike the model of Garnick (1992), due to our coinfection class we do not see competitive exclusion here, and we also do not restrict the host populations to remain constant. The only manner in which the parasites may affect therefore each other is through the increased death rate that they induce in infected hosts, reducing the susceptible population for the other parasite.

Here we check the stability of the steady state when each parasite is absent. For the second parasite we see from equations 2.1.1 and 2.2.2 that, investigating the Jacobian of the subsystem of the second parasite only, with the first parasite at a stable equilibrium, we require an eigenvalue of

$$J_2 = \begin{pmatrix} \beta_2^{22} S_2^\dagger - \beta_1^{21} I_{11}^\dagger - \beta_1^{22} I_{21}^\dagger - d_2 - \gamma_{22} & 0 \\ \beta_1^{22} I_{21}^\dagger + \beta_2^{22} I_{21}^\dagger & \frac{\beta_1^{22}+\beta_2^{22}}{2} S_2^\dagger + \beta_1^{22} I_{21}^\dagger - d_2 - \omega_2 \end{pmatrix}$$

to be positive in order for the second-parasite-free steady state to be unstable. If we assume that intra- and inter-species infection rates of both parasites are similar, i.e. $\beta_1^{21} \approx \beta_1^{22} \approx \beta_2^{22}$, and coinfection does not significantly increase mortality, i.e. $\gamma_{22} \approx \psi_2$, we get the condition that the system is unstable, and hence the second parasite can invade, if

$$S_2^\dagger - I_{11}^\dagger - I_{21}^\dagger > \frac{d_2 + \gamma_{22}}{\beta_2^{22}}. \tag{2.3.10}$$

This implies that the second parasite will always survive in a susceptible host population large enough, and in such a population will never be driven to extinction. Part of the implication of this is that the presence of the second parasite depends on the death rate induced by the first, affecting the susceptible population, which results in indirect competition. In addition, an increase in mortality due to coinfection could have a significant effect here, as

could decreased transmission rates for coinfection (a likely possibility, given that generalism will probably incur transmission costs).

However, for our assumptions, as long as the equilibrium value of hosts available to the second parasite is greater than the constant on the right-hand side of equation 2.3.10, then the parasite will persist. This equilibrium will obviously be affected by the presence of the first parasite, changing the survival chances of the second through altered mortality rates, and hence susceptible host equilibrium values.

In comparison, repeating this with the first parasite yields a Jacobian given by

$$J_1 = \begin{pmatrix} J(1,1) & \beta_1^{12} S_1^* & 0 \\ \beta_1^{21} S_2^* & J(2,2) & 0 \\ 0 & \beta_1^{22} I_{22}^* + \beta_2^{22} I_{22}^* & J(3,3) \end{pmatrix},$$

where

$$\begin{aligned} J(1,1) &= \beta_1^{11} S_1^* - d_1 - \gamma_{11} - \beta_2^{12} I_{22}^*, \\ J(2,2) &= \beta_1^{22} S_2^* - d_2 - \gamma_{21} - \beta_2^{22} I_{22}^*, \\ J(3,3) &= \frac{\beta_1^{22} + \beta_2^{22}}{2} S_2^* + \beta_2^{22} I_{22}^* - d_2 - \omega_2. \end{aligned}$$

While the eigenvalues for this are not as clear as the solution for the second parasite above, it must be noted again that the first parasite will always be able to invade if the number of available hosts is big enough and the rate of infection is faster than the death rates. Again, this much seems reasonable; as parasites do not interact, save through indirect competition, they are able to coexist if that competition is low enough and they have the potential to exist in the absence of their competitor.

We see in figure 2-2 that a specialist parasite species only out-competes a generalist for a select section of parameter values, and may even suffer extinction, while the generalist does not become extinct for any of the parameter values presented. It is also important to note that any equilibrium we reach is independent of the initial conditions (provided all species are initially present), and only one equilibrium exists for each set of input parameters (Garnick, 1992). From our results, however, we are not able to compare whether host-parasite networks should be either more nested or anti-nested than expected due to chance alone. We therefore investigate the values of R_0

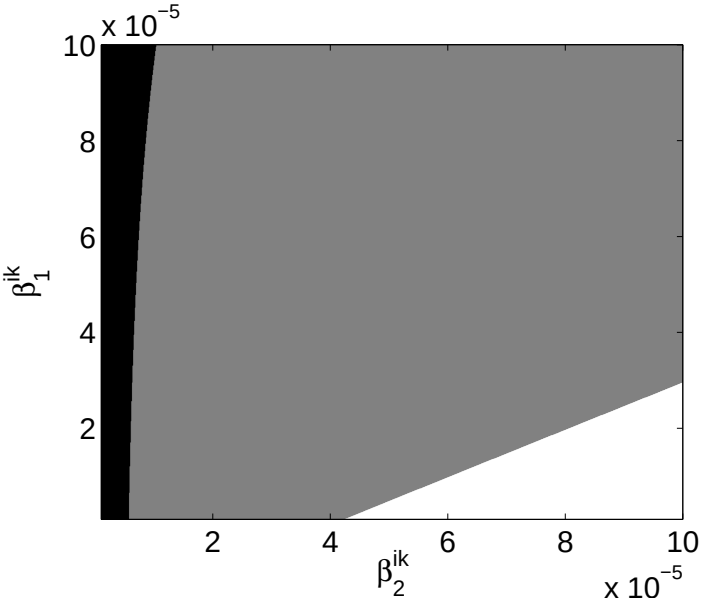


Figure 2-2: Plot of the regions of transmission parameters for which the specialist out-competes the generalist (white), coexists with the generalist (grey) and is driven to extinction (black). Note that the generalist is never driven to extinction if it would survive in the absence of the specialist.

in a different manner.

2.3.1 Comparative R_0

Here we investigate two cases, when a parasite species is introduced to a system with a specialist parasite already present at equilibrium, and when a generalist parasite species is already present. The aim of this is to discover whether certain parasites are favoured in each scenario, leading to the structure described in figure 2-1. If a specialist parasite is present, we expect that R_0 will be higher for a generalist parasite or a specialist parasite in the same host than for a specialist parasite in the opposite host. If a generalist parasite is present, we expect that R_0 will be higher for a specialist parasite in either host than for a generalist. We calculate the R_0 values for each of the cases described above, and discover that there is a very select range of transmission parameters for which this is the case, but we certainly do not see a pervasive pattern promoting nestedness.

A parasite entering a system in which a specialist is already present could correspond to three of the above scenarios for the first parasite entering a system; with one host and one parasite (i.e. a specialist introduced to the

opposite host, equation 2.2.4), a one host, two parasite system (i.e. a specialist introduced to the same host as the parasite already present, equation 2.2.6) and the full system (of which only the NGM is given above, i.e. a generalist entering the system, equation 2.2.3). Note that in our model the value of R_0 for a specialist entering the same host as another specialist is always lower than that for a specialist entering the opposite host. This is due to the costs that parasitism incurs on hosts in our model, reducing the potential size of the host population. This means that we are unlikely to see specialization asymmetry of the form given in figure 1-1(b) here.

For the case where we introduce a parasite to a system in which there is already a generalist, we see that it corresponds to the full system for the second parasite (i.e. a specialist introduced to a system containing a generalist, equation 2.2.7) or to the addition of a second generalist parasite, for which we give the NGM below

$$K = \begin{pmatrix} \frac{\beta_2^{11} S_1^*}{k_1} & \frac{\beta_2^{12} S_1^*}{k_2} & 0 & 0 \\ \frac{\beta_2^{22} S_2^*}{k_1} & \frac{\beta_2^{21} S_2^*}{k_2} & 0 & 0 \\ \frac{\beta_2^{11} I_{11}^*}{k_1} & \frac{\beta_2^{12} I_{11}^*}{k_1} & \frac{g_{11}}{d1+\psi_1} & \frac{g_{12}}{d2+\psi_2} \\ \frac{\beta_2^{21} I_{11}^*}{k_1} & \frac{\beta_2^{22} I_{21}^*}{k_2} & \frac{g_{21}}{d1+\psi_1} & \frac{g_{22}}{d2+\psi_2} \end{pmatrix},$$

where

$$\begin{aligned} k_1 &= d_1 + \gamma_{12} + \beta_1^{11} I_{11}^* + \beta_1^{12} I_{21}^*, \\ k_2 &= d_2 + \gamma_{22} + \beta_1^{21} I_{11}^* + \beta_1^{22} I_{21}^*, \\ g_{mn} &= \frac{(\beta_1^{mn} + \beta_2^{mn}) S_m^*}{2} + \beta_2^{mn} I_{m1}^* + \beta_1^{mn} I_{m2}^*. \end{aligned}$$

Comparing values for the various basic reproductive numbers discussed above, we obtain figure 2-3. This figure demonstrates the cost levels that would have to be exacted on transmission rates in order to ensure that a specialist would have a higher basic reproductive number than a generalist, whether entering a system containing a specialist or a generalist species already. We see this is roughly 0.5, from the slope of figure 2-3. This implies that, to be a generalist rather than a specialist, the cost to transmission rates should be that they are less than halved. This is perhaps unsurprising, given that the pool of susceptible individuals doubles in size if one is to become a generalist. We would imagine that this result generalises to larger systems,

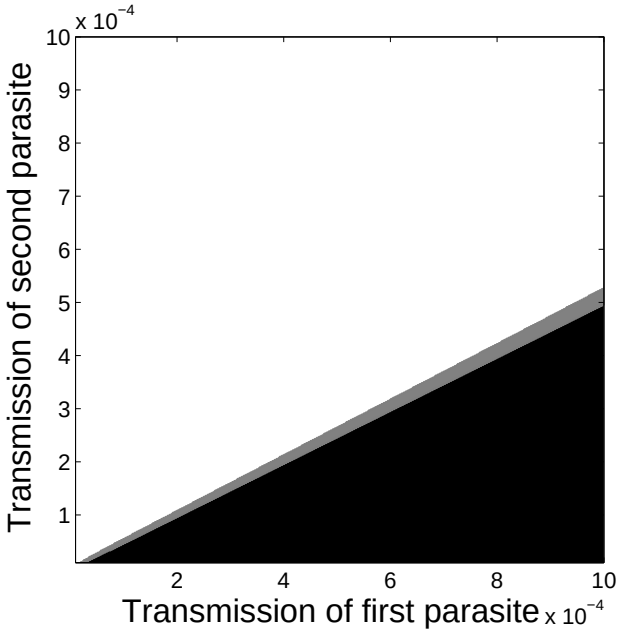


Figure 2-3: Transmission parameters which result in invasion of a specialist or a generalist species. We investigate the basic reproductive number of a second parasite when it is introduced to a system with one specialist parasite and two host species at equilibrium, and separately with one generalist parasite and two host species at equilibrium. The black area denotes transmission parameters for which R_0 is greater for the second parasite as a specialist (in either host species) than as a generalist, whichever system it is introduced to. The white area denotes transmission parameters for which R_0 is greater for the second parasite as a generalist than as a specialist (in either host species), whichever system it is introduced to. In the grey region, R_0 is greater for a generalist when the parasite is entering a system with a specialist, and for a specialist when the parasite is entering a system with a generalist. This region, therefore, sees transmission parameters that promote the structure seen in figure 2-1. Note that if the parasite is a specialist then it is presumed to have the same transmission parameter as the first parasite, while if it is a generalist then its transmission parameter is given on the y-axis. Also, intra- and inter-species transmission rates are identical.

although no work is done on this here.

We see almost no overlap in which type of species is more successful; if the cost to generalism is such that rates are less than halved, then a generalist is more likely to enter a system containing a specialist, but also a generalist is more likely to enter a system containing a generalist. The same applies if transmission rates are more than halved due to generalism; i.e. specialists always win. The region in which we always see promotion of a nested structure is very small, and seems unlikely to be the driving force behind nested patterns

in host-parasite networks. Although this could lead to patterns of nestedness and anti-nestedness, as observed empirically, it does imply that parasites should either always be specialists or always be generalists, depending on the transmission cost to generalism. This is clearly not the case, as the specificity of parasites is not identical across species. This is in part due to species evolution, a factor not considered here. Hence, in the following chapters as we are forced to look for new methods to explain nestedness mathematically, we begin to consider evolutionary dynamics.

2.4 Conclusion

Both parasites in this model depend implicitly on the mortality which the other introduces, and the rate at which they infect the host population. From the perspective of species richness, only the increased mortality is noticeable. Accordingly, if both hosts were identical in all other respects, it would seem likely that both parasites would preferentially infect a species-poor host (these hosts would lower effective death rates) or become generalists (unless the cost were to be too high).

We conclude that there is an essential aspect to the problem that the model is missing, as it does not promote patterns of nestedness. Given an identical, linear cost to generalism across parasite species, the cost is either so great as to always promote specialists, or so little as to always promote generalists. However, if a specialist attacks a host which contains other specialists, then the host's defences would be less likely to cope, as they would be focused on other parasites. If many specialist parasite species did this, then each individual host might be less likely to be able to defend itself against multiple parasite species simultaneously. Also, if a specialist attacked a host which contained generalist species, then its defences could be more adaptable, as generalists often have a non-specific attack mechanism. This could make it easier for the host to adapt and target the specialist. This verbal reasoning leads us to propose a physiological model involving the trade-off of resources. In this model the potential rewards are considered for specialists and generalists, and the environments in which they are found. In addition, the reaction of host species could be of great importance, and must certainly be considered. Both host and parasite species are therefore allowed to evolve, in order to adapt to an ever-changing system.

CHAPTER 3

Two-Dimensional Host-Parasite System

Our previous model investigated a system with two hosts and two parasites, as a starting point for a higher-dimensional system. In this system, one host was infected by both parasites (making it comparatively species rich), while the other contained only one of the parasites (making that parasite a comparative generalist). This set of interactions was fixed, allowing us to predict the circumstances under which one or the other of the parasite species would thrive more. We did not discover any evidence that a nested architecture would be promoted in such a system, so here we allow the interactions to evolve, and include a trade-off between parasite attack strategies and host defensive strategies on each species.

Vázquez et al. (2005) has constructed a null model based on host abundance to account for specialization asymmetry, yet there may be many more explanations for the link between the specificity of a parasite and the parasite species-richness of the hosts that it infects (Lewinsohn et al., 2006; Poulin, 2007a). Species abundance does not, for example, explain the frequent occurrence of anti-nestedness, another non-random pattern of ecological networks (Joppa et al., 2010). Further explanations for nestedness in networks include complementarity (Rezende et al., 2007), based on phenotypic matching between species, and competitive load (Bastolla et al., 2009), based on a new species entering a network targeting a host with less competition provided by resident parasite species. Nestedness could also be due to body size in a

cascade hierarchy (Woodward et al., 2005), although this again fails to explain patterns of anti-nestedness in host-parasite networks, or it could even be due to a combination of the above, such as body size and abundance acting together (Montoya et al., 2006).

Another possible driving force behind the link between specificity and species richness is related to the levels of defence that a host exhibits. For example, avian fleas with different levels of specificity target hosts with different levels of T-cell mediated immune response (Møller et al., 2005). Generalist parasites target hosts with weak levels of immune response, while parasites with fewer host species exploit those with both strong and weak immune responses. This also has an effect on the parasite species-richness of host species, with hosts with stronger immune responses being parasitized by a greater number of species. In this instance, then, the specificity of parasites and specialization asymmetry appears to be related to the host immune response (Møller et al., 2005). Here, this idea is turned around slightly, but the concept of a relationship between host response and specificity is maintained.

It is becoming increasingly apparent that the coevolution of species is an important driving force in host-parasite relationships (Best et al., 2009). Best et al. (2010) show the importance of such coevolutionary ecological feedbacks in the adaption of species to one another, and the effects that this has on the evolution and branching of resistance and infectivity traits. This coevolution can lead to more complicated and interesting dynamics than simple evolution only, such as cycles in levels of virulence and resistance (Sasaki, 2000), and is known to influence the strength of interspecies interactions (Kopp and Gavrilets, 2006). It is also widely acknowledged that trade-offs in resource allocation are responsible for much evolutionary drive in parasites, such as the link between virulence and transmission (May and Anderson, 1983). In this chapter it is postulated that the link between specificity and richness may be driven by the coevolution of both hosts and parasites when balancing the allocation of resources (see Poulin and Morand, 2004). These resources are devoted in different degrees to interactions with one species versus another, either for infection or for preventing infection.

As an example, the influenza virus binds to cell-surface oligosaccharides via a sialic acid receptor. The receptor type may have one of two conformations: Neu 5Acα(2,3)-Gal or Neu5Acα(2,6)-Gal. A host species may have one of the two or both (Cobey et al., 2010), but the virus must adapt to

one linkage type at the expense of the other. Other examples include phenotypic trait matching, such as the shaping of mouthparts of ectoparasites to match the skin surface of their host (Graham et al., 2009) or insect proboscis shape matching plant nectar holder size and shape in mutualistic pollinator networks (Vázquez et al., 2009a). From a host perspective, a number of examples of predator- or parasite-mediated trade-offs in resource allocation can be found, focused mainly on changes in spatial usage or diet. Thiemann and Wassersug (2000) discuss the reduction of activity by *Rana* tadpoles in response to increased exposure to predators. This decrease in activity results in an increase in infection by trematodes. A similar scenario is also played out by freshwater snails (Rigby and Jokela, 2000). Baboons are known to eat the leaves and berries of a particular shrub to kill off internal schistosoma in areas of high infection risk (Lozano, 1991), demonstrating a behavioural response to an increase in parasite density. Grasshoppers jump at an intermediate height, to reduce the threat of predation from birds from above as well as small mammals and lizards from below (Pitt, 1999). Lastly, reindeer and other herd animals often group in a reaction to parasitism by biting flies, which reduces their chances of attack, but does lead to an increased risk of exposure to other pathogens which rely on host density for transmission (Hart, 1988).

Using the above concepts, the following is proposed: if an infection is more prevalent, then a host will have a higher likelihood of coming into contact with it and adapting to fight it, allocating a greater amount of resource to this and increasing its immune response. A host has, however, limited resources with which to do so (Poitrineau et al., 2003; Poulin and Morand, 2004). Similarly, a parasite may infect multiple hosts. It will, however, be better adapted to infect some than others, and again there will be a trade-off in terms of its efficiency in infecting a host species (see Poulin, 1998b). It is therefore assumed that both host and parasite species trade off their resources between those species that they target.

This trade-off aims to incorporate all of the ideas discussed above. A more abundant species will provide more available hosts for a parasite. A lower competitive load will encourage infection of that host as an untapped resource. Lastly, complementarity will ensure that a species will target another with complimentary trait values (parasites will infect hosts which are more vulnerable to them, while hosts will protect themselves against parasites

that are more of a threat).

However, the trade-off between two attack or defence mechanisms may not always be clear-cut, as discussed by Maleck and Dietrich (1999) on the responses of plants; systemic acquired resistance interacts with the wound response pathway to parasitism through a complex system of interactions, without the presence of a clear trade-off. Here we assume the most mathematically simple trade-off, but include the caveat that most trade-offs will be far more complex.

With this in mind, we create a toy model here which investigates the coevolution of trade-offs in a dynamical manner, for a four-species system containing two species of each type; hosts and parasites. This may be thought of as a cluster of species forming a 'compartment' in a larger food web, and hence more general results may be inferred from the results obtained (see Joppa et al., 2009). Although this is simply a toy problem, figure 1-1 gives an indication of what specialization asymmetry might look like in this case, and hence the patterns we might expect the model to show for nestedness and anti-nestedness in a larger system. It is important to stress that in order to fully understand the influence of these trade-offs on nestedness, the investigation of a larger system is necessary (see Chapter 4).

We outline a model describing the system and its mathematical motivation, following this with a brief analysis and an investigation of the coevolutionary dynamics. The results are compared to a previous null model based on abundance (Vázquez et al., 2005), in order to demonstrate the compatibility of this theory with our model.

3.1 The Model

A standard susceptible-infected system is assumed, with two species of hosts and two of parasites. This model has the potential for both specialist and generalist parasites (in one or both hosts respectively) as well as species-poor and species-rich hosts (containing neither parasite, one only or both). S_i refers to susceptibles of host type i, while I_{ij} refers to infecteds of host type

i with parasite type j, where in this instance $i, j \in \mathbb{N}_2$. We then have

$$\begin{aligned}\frac{dS_i}{dt} =&\alpha_i M_i - \sum_{j\in\mathbb{N}_2} a_{ij}c_{ij}S_iF_j^i - \omega_i S_i M_i,\\ \frac{dI_{ij}}{dt} =&a_{ij}c_{ij}S_iF_j^i - \gamma_{ij}I_{ij} - \omega_i I_{ij}M_i.\end{aligned} \tag{3.1.1}$$

The model contains per-capita birth (α_i) and density-dependent death (ω_i) rates dependent on the host species i, as well as infection-related death; death rate γ_{ij} of host species i due to parasite species j. $M_i = S_i + I_{i1} + I_{i2}$ represents the total population size of host species i.

The *maximum* force of infection F_j^i of parasite species j on host species i is given by

$$F_j^i = \sum_{k\in\mathbb{N}_2} \beta_j^{ik} I_{kj},$$

where β_j^{ik} is the pairwise potential infectious contact rate for the transfer of parasite j from host k to host i. In our model, the *actual* force of infection G_j^i is assumed to be moderated by the strategies adopted by the parasite j and the host i. It is given by

$$G_j^i = a_{ij}c_{ij}F_j^i = a_{ij}c_{ij}(\beta_j^{i1}I_{1j} + \beta_j^{i2}I_{2j}), \tag{3.1.2}$$

with $0 \le a_{ij} \le 1$, $0 \le c_{ij} \le 1$. Here a_{ij} is a parasite-related trait defining the relative probability of success of parasite j's attack on host i, and c_{ij} is a host-related trait defining the relative probability of failure of host i's defence against parasite j. All else being equal, parasites benefit from values of a_{ij} that are as high as possible, while hosts benefit from values of c_{ij} that are as low as possible.

We assume, however, that each parasite species j has a fixed amount of resource to allocate to infection, and that there is therefore a trade-off between the strength a_{1j} of its attack against host 1 and the strength a_{2j} of its attack against host 2. This trade-off is assumed to be a decreasing function, which is species specific and is not dependent upon the population or environment. A host species i, meanwhile, varies strategy c_{ij} in order to reduce transmission of parasite j, and a similar trade-off is presumed.

Transmission of infection to a susceptible host then depends on the actual force of infection, a measure of both the propensity of the parasite to infect that host, as well as the host's propensity to defend itself against the parasite.

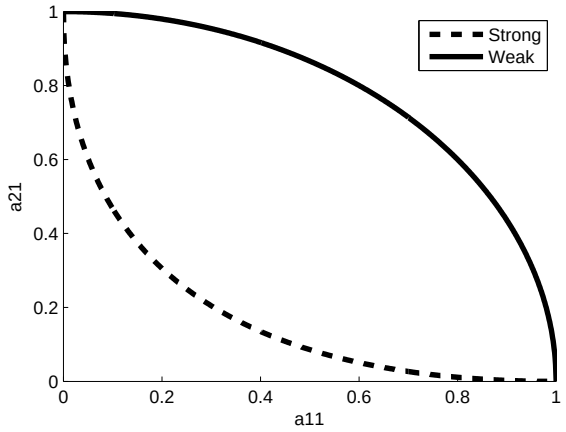


Figure 3-1: Examples of trade-off shapes for trait values of parasite species 1, where the trade-off is either strong ($\theta_1 = 0.5$) or weak ($\theta_1 = 2$)

In reality, it is difficult to determine the shapes such trade-offs take (Best et al., 2009), and these can affect aspects such as the generation of diversity (Bastolla et al., 2009). A general trade-off shape therefore allows for a greater understanding of different possible evolutionary outcomes (Kisdi, 2006). Points at which an evolutionarily stable strategy (ESS) may exhibit evolutionary branching also often depend on the nature of the trade-off function presumed; more specifically, whether it is concave or convex, and the extent of this (Kisdi, 2006). Levins' fitness set approach (Rueffler et al., 2006) has previously influenced intuitive thoughts on the effects of these shapes under normal evolutionary conditions, with a generalist expected if a trade-off is convex, or weak, and either specialist expected if a trade-off is concave, or strong.

The trade-off shape is determined here by a species-specific power (θ_j for parasite species j and ϕ_i for host species i), where

$$a_{1j}^{\theta_j} + a_{2j}^{\theta_j} = 1 \quad \text{and} \quad c_{i1}^{\phi_i} + c_{i2}^{\phi_i} = 1.$$

For parasite species j, $\theta_j < 1$ implies a strong trade-off, and $\theta_j > 1$ implies a weak trade-off (see figure 3-1). A parasite is a perfect generalist if $a_{1j} = a_{2j} = (0.5)^{\frac{1}{\theta_j}}$, which is henceforth termed the *neutral point*. It is a complete specialist if $a_{1j} = 0$ and $a_{2j} = 1$, or *vice versa*. This is similar for host species. Note that the tendency in nature is for trade-offs to be strong, not weak (Rueffler et al., 2006).

The second derivative of the trade-off function, the degree to which it is concave or convex, can have an enormous effect on the possibility of evolu-

tionary branching (see Kisdi, 2006). The sign of this depends wholly upon the value of the species-specific trade-off shape, and so the system will exhibit very different behaviour around this point. The more concave a function is, the more likely the system is to exhibit branching (Kisdi, 2006). Note that the second derivative is maximized (either positive or negative depending on the trade-off shape) for parasite j at $a_{1j} = (0.5)^{\frac{1}{\theta_j}}$, where

$$\frac{d^2 a_{2j}}{da_{1j}^2} = (\theta_j - 1),$$

and similarly for host species. This affects the direction in which trait-values mutate, which will be discussed in greater detail in the following sections.

3.2 Analysis

This model is analysed using adaptive dynamics. This assumes rare mutants with phenotypic traits that are marginally different from those of residents, which may then invade the population if their growth rate is positive in an equilibrium environment (Dercole et al., 2003). This approach assumes clonal reproduction, but the results still hold for random mating in monomorphic diploid populations with polygenic traits if mutations are rare with small phenotypic effect (Law et al., 2001; Rueffler et al., 2006). The growth rate of a mutant phenotype while rare is termed the invasion fitness, and is important in determining whether that mutant may invade, and potentially replace, a resident (de Mazancourt and Dieckmann, 2004). The population evolves in the direction of the fitness gradient (the change in the invasion fitness with respect to change in the mutant trait value) as successive mutations occur and then spread through the population (de Mazancourt and Dieckmann, 2004). The invasion fitness can then be used to discover singularities, where the fitness gradient of any local mutant is zero, and to investigate the nature of these singularities (Geritz et al., 1998). Examples of the derivation of the invasion conditions for both parasites and hosts are given below for a system without coinfection or recovery.

3.2.1 Parasite invasion conditions

In determining the invasion fitness of a mutant parasite in the population, here a mutant I'_{i1} with strategy (a'_{i1}) of parasite 1, with the resident population

at a stable, non-trivial equilibrium ($S_i = S_i^*$, $I_{ij} = I_{ij}^*$), the steady state is determined below for all i, j as

$$\begin{aligned}\frac{dS_i^*}{dt} =& \alpha_i M_i^* - \sum_{j\in\mathbb{N}_2} a_{ij}c_{ij}S_i^*F_j^{i*} - \omega_i S_i^* M_i^* = 0,\\ \frac{dI_{ij}^*}{dt} =& a_{ij}c_{ij}S_i^*F_j^{i*} - \gamma_{ij}I_{ij}^* - \omega_i I_{ij}^* M_i^* = 0,\end{aligned} \tag{3.2.3}$$

and the linearized dynamics of the mutant are expressed as follows:

$$\frac{dI_{i1}'}{dt} = a_{i1}'c_{i1}S_i^*F_1^{i'} - \gamma_{i1}I_{i1}' - \omega_i I_{i1}' M_i^*. \tag{3.2.4}$$

The resident is at a stable equilibrium, which implies that the Jacobian of the system can be split into the original dynamics and a separate submatrix determined by the dynamics of the mutant, given by $\boldsymbol{A}_1$ (Diekmann and Heesterbeek, 2000; Hurford et al., 2010; van den Driessche and Watmough, 2002).

Defining $\delta_{11} = a_{11}' - a_{11}$ and $\delta_{21} = a_{21}' - a_{21}$, both small, we get

$$\boldsymbol{A}_1 = \begin{pmatrix} A_1(1,1) & A_1(1,2) \\ A_1(2,1) & A_1(2,2) \end{pmatrix}, \tag{3.2.5}$$

where the equilibrium of the system is used to guarantee that

$$I_{i1}^*(\gamma_{i1} + \omega_i M_i^*) = a_{i1}c_{i1}S_i^*F_1^{i*},$$

and hence, for $n \in \mathbb{N}_2$,

$$\begin{aligned}\boldsymbol{A}_1(n,n)I_{n1}^* &= \delta_{n1}c_{n1}S_n^*\beta_1^{nn}I_{n1}^* - a_{n1}c_{n1}S_n^*\beta_1^{n(n-1)}I_{(n-1)1}^*,\\ \boldsymbol{A}_1(n,n-1) &= (\delta_{n1} + a_{n1})c_{n1}S_n^*\beta_1^{n(n-1)}.\end{aligned}$$

The stability of $\boldsymbol{A}_1$ is then used to indicate the potential for invasion. As it is difficult to interpret anything useful from the eigenvalues, a sign-equivalent proxy can be found for the growth rate of the mutant by investigating the trace and determinant of $\boldsymbol{A}_1$. The determinant is given by

$$I_{11}^*I_{21}^*\det(\boldsymbol{A}_1) = -c_{11}c_{21}S_1^*S_2^*u_1(\delta_{11}, a_{11}, \delta_{21}, a_{21}),$$

where, to leading order of δ,

$$\begin{aligned} u_1(\delta_{11}, a_{11}, \delta_{21}, a_{21}) =& \beta_1^{12}\beta_1^{21} I_{11}^* I_{21}^* (\delta_{11} a_{21} + a_{11}\delta_{21}) \\ &+ \beta_1^{21}\beta_1^{11} (I_{11}^*)^2 \delta_{11} a_{21} \\ &+ \beta_1^{12}\beta_1^{22} (I_{21}^*)^2 a_{11}\delta_{21}. \end{aligned} \tag{3.2.6}$$

When $a'_{11} = a_{11}$, then $\mathrm{tr}(\boldsymbol{A}_1) < 0$ and $\det(\boldsymbol{A}_1) = 0$, as $\delta_{n1} = 0$. For the small perturbations resulting from mutation we can therefore rely on the determinant condition for stability analysis, as the trace will remain negative. The cases for $I_{11}^* = 0$ and $I_{21}^* = 0$ are discussed later. The determinant will be negative, and hence the system is unstable and the mutant invades, if

$$u_1(\delta_{11}, a_{11}, \delta_{21}, a_{21}) > 0. \tag{3.2.7}$$

Interpreting this condition, invasion of a mutant parasite can be seen to be driven by the following; the first term is due to inter-species transmission, and is minimized at the neutral point $a_{11} = a_{21}$, hence driving towards generalism. This is due to an increased invasion probability when a mutant is investing more equally than the resident, as $(\delta_{11}a_{21} + a_{11}\delta_{21})$ is larger. When $a_{11} = a_{21}$ then this term is zero, and no possible mutant can invade due to this. When $a_{11} < (0.5)^{\frac{1}{\theta_1}}$, this implies that $a_{21} > (0.5)^{\frac{1}{\theta_1}}$. Hence we require a mutant with a larger value of a_{11} to promote invasion, and *vice versa* for $a_{11} > (0.5)^{\frac{1}{\theta_1}}$. So this term stabilizes at and attracts to the neutral point, promoting generalism.

The final two terms compare the use of the two available hosts. For example, if transmission due to host 1 ($\beta_1^{21}\beta_1^{11}(I_{11}^*)^2$) is high, then a mutant with larger a'_{11} will invade, ensuring host 1 is utilized. These terms demonstrate an increased invasion potential if the mutant increases infection of the species with higher infection rates ($\beta_1^{21}\beta_1^{11}$ compared to $\beta_1^{12}\beta_1^{22}$) and on which the resident relies more (whether I_{11}^* is greater or less than I_{21}^*), as δ_{11} and δ_{21} have opposite signs. This ensures that the mutant is spread as much as possible to susceptible hosts.

Note that the case where either $I_{11}^* = 0$ or $I_{21}^* = 0$ sees the parasite shy away from a completely protected host. These points are only obtainable, for a four-species system where the maximum force of infection is not zero, if the actual force of infection is zero, i.e. $a_{11}c_{11} = 0$ or $a_{21}c_{21} = 0$ as appropriate.

For the case where $I_{11}^* = 0$, the submatrix of the Jacobian is given by

$$\boldsymbol{A}_1 = \begin{pmatrix} -\gamma_{11} - \omega_{11}(S_1^* + I_{12}^*) & 0 \\ (\delta_{21} + a_{21})c_{21}S_2^*\beta_1^{21} & \delta_{21}c_{21}S_2^*\beta_1^{22} \end{pmatrix}. \tag{3.2.8}$$

The mutant can thus invade only if $\delta_{21} > 0$, i.e. if $a'_{11} < a_{11}$. A similar situation arises for $I_{21}^* = 0$. If both I_{11}^* and $I_{21}^* = 0$ then the system is at a trivial equilibrium, in which case no mutant can invade.

3.2.2 Host invasion conditions

Similarly to the parasite case above, a mutant population (S'_1, I'_{1j}) with trait value c'_{1j} of host 1 is introduced at low densities to the resident population at equilibrium ($S_i = S_i^*$, $I_{ij} = I_{ij}^*$). The steady state of the resident system and dynamics of the mutant subsystem are given by

$$\begin{aligned}
\frac{dS_i^*}{dt} =& \alpha_i M_i^* - \sum_{j\in\mathbb{N}_2} a_{ij}c_{ij}S_i^*F_j^{i*} - \omega_i S_i^* M_i^* = 0,\\
\frac{dI_{ij}^*}{dt} =& a_{ij}c_{ij}S_i^*F_j^{i*} - \gamma_{ij}I_{ij}^* - \omega_i I_{ij}^* M_i^* = 0,\\
\frac{dS'_1}{dt} =& \alpha_1 M'_1 - a_{11}c'_{11}S'_1F_1^{1*} - a_{12}c'_{12}S'_1F_2^{1*} - \omega_1 S'_1 M_1^*,\\
\frac{dI'_{1j}}{dt} =& a_{1j}c'_{1j}S'_1F_j^{1*} - \gamma_{1j}I'_{1j} - \omega_1 I'_{1j}M_1^*.
\end{aligned}$$

The equilibrium conditions are taken from equation 6.1.1, and we define $\eta_{11} = c'_{11} - c_{11}$ and $\eta_{12} = c'_{12} - c_{12}$, both small. Again we investigate the submatrix of the Jacobian, given by

$$\boldsymbol{C}_1 = \begin{pmatrix} C_1(1,1) & \alpha_1 & \alpha_1 \\ a_{11}(\eta_{11} + c_{11})F_1^{1*} & -\frac{a_{11}c_{11}S_1^*F_1^{1*}}{I_{11}^*} & 0 \\ a_{12}(\eta_{12} + c_{12})F_2^{1*} & 0 & -\frac{a_{12}c_{12}S_1^*F_2^{1*}}{I_{12}^*} \end{pmatrix}, \tag{3.2.9}$$

where

$$\boldsymbol{C}_1(1,1) = \alpha_1\left(1 - \frac{M_1^*}{S_1^*}\right) - a_{11}\eta_{11}F_1^{1*} - a_{12}\eta_{12}F_2^{1*}.$$

Now, as the subsystem is three-dimensional, the eigenvalues are given as solutions to an equation of the form $\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3 = 0$, where

$$\begin{aligned}
b_1 =& -\operatorname{tr}(\boldsymbol{C}_1),\\
2b_2 =& \operatorname{tr}^2(\boldsymbol{C}_1) - \operatorname{tr}(\boldsymbol{C}_1^2),\\
b_3 =& -\det(\boldsymbol{C}_1).
\end{aligned}$$

For stability, it is required that $b_1, b_2, b_3 \geq 0$ and $b_1 b_2 \geq b_3$, while instability occurs if either of these inequalities is violated. When $c'_{11} = c_{11}$, both b_1 and b_2 are positive and $O(1)$, as $\eta = 0$. On the other hand,

$$b_3 = -\frac{1}{I^*_{11} I^*_{12}} a_{11} a_{12} S^*_1 F^{1*}_1 F^{1*}_2 w_1(\eta_{11}, c_{11}, \eta_{12}, c_{12}),$$

where

$$\begin{aligned} w_1(\eta_{11}, c_{11}, \eta_{12}, c_{12}) =& c_{12}\eta_{11}\alpha_1 I^*_{11} + c_{11}\eta_{12}\alpha_1 I^*_{12} \\ & - c_{11}c_{12}a_{11}S^*_1 F^{1*}_1 \eta_{11} \\ & - c_{11}c_{12}a_{12}S^*_1 F^{1*}_2 \eta_{12}. \end{aligned} \tag{3.2.10}$$

Now this is $O(\eta)$, and is zero when $c'_{11} = c_{11}$, hence $b_1 b_2 > b_3$. The criteria for *instability* (and invasion) when mutations are small can therefore be reduced to

$$w_1(\eta_{11}, c_{11}, \eta_{12}, c_{12}) > 0. \tag{3.2.11}$$

Interpreting this condition, it can be seen that mutant invasion depends on a balance of terms. The first two terms from equation 3.2.10 push the host towards a generalized defence. For example, a particularly large value of c_{11} makes the second term likely to be larger, and hence a mutant with a smaller trait value c'_{11} will invade, to ensure that $\eta_{12} > 0$.

The final two terms decrease in importance ($c_{11}c_{12}$ decreases), compared to the initial terms, as the host specializes. If the pressure from parasite 1 is higher (the actual force of infection is higher) then the first of these terms will be larger. A host mutant with a smaller trait value will therefore invade, in order to make η_{11} negative and the sum of the final two terms positive. In this way the mutant host protects itself against parasite 1 to a greater extent. These terms then account for the pressure that a parasite places on the host, and the host's reaction to this.

The case where either I^*_{11} or I^*_{12} is zero, which again occurs if $a_{11}c_{11} = 0$ or $a_{12}c_{12} = 0$ as appropriate, shows that a host will not defend itself against a non-threatening parasite, and if the system is at a trivial equilibrium, then no mutant may invade. For example, if $I^*_{11} = 0$, then equation 3.2.9 is given by

$$\boldsymbol{C}_1 = \begin{pmatrix} \boldsymbol{C}_1(1,1) & \alpha_1 & \alpha_1 \\ 0 & -\gamma_{11} - \omega_1 M^*_1 & 0 \\ a_{12}c'_{12}F^{1*}_2 & 0 & -\frac{a_{12}c_{12}S^*_1 F^{1*}_2}{I^*_{12}} \end{pmatrix}, \tag{3.2.12}$$

where $\boldsymbol{C}_1(1,1) = \alpha_1\left(1 - \frac{M_1^*}{S_1^*}\right) - a_{12}\eta_{12}F_2^{1*}$. In this case, a stronger than necessary condition is that a mutant will always invade if $a_{12}\eta_{12}F_2^{1*} < 0$, i.e. if $c'_{11} > c_{11}$. A similar situation arises for $I_{12}^* = 0$. Note that the cases for mutants of parasite and host species 2 are similar to the above.

The conditions for invasion can be independently verified using the next-generation tools outlined in van den Driessche and Watmough (2002). For $\rho(\boldsymbol{M})$ defined to be the spectral radius of matrix $\boldsymbol{M}$, and $s(\boldsymbol{M})$ the spectral bound, the Jacobian $\boldsymbol{C}_1$ is decomposed such that

$$\boldsymbol{C}_1 = \boldsymbol{F}_1 - \boldsymbol{V}_1, \tag{3.2.13}$$

for $\boldsymbol{F}_1$ and $\boldsymbol{V}_1$ satisfying $s(-\boldsymbol{V}_1) < 0$, $\boldsymbol{V}_1^{-1} \geq 0$ and $\boldsymbol{F}_1 \geq 0$. $\boldsymbol{F}_1$ and $\boldsymbol{V}_1$ are taken as matrices representing the appearance and disappearance of individuals from the system respectively. The invasion condition is then given by

$$\rho(\boldsymbol{F}_1\boldsymbol{V}_1^{-1}) > 1, \tag{3.2.14}$$

For further details see van den Driessche and Watmough (2002) or Hurford et al. (2010). In this case, we take

$$\boldsymbol{F}_1 = \begin{pmatrix} \alpha_1 & \alpha_1 & \alpha_1 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{3.2.15}$$

and

$$\boldsymbol{V}_1 = \begin{pmatrix} \boldsymbol{V}_1(1,1) & 0 & 0 \\ -a_{11}(\eta_{11} + c_{11})F_1^{1*} & \frac{a_{11}c_{11}S_{11}^*F_1^{1*}}{I_{11}^*} & 0 \\ -a_{12}(\eta_{12} + c_{12})F_2^{1*} & 0 & \frac{a_{12}c_{12}S_{11}^*F_2^{1*}}{I_{12}^*} \end{pmatrix}, \tag{3.2.16}$$

where $\boldsymbol{V}_1(1,1) = \alpha_1\frac{M_1^*}{S_1^*} + a_{11}\eta_{11}F_1^{1*} + a_{12}\eta_{12}F_2^{1*}$. After some straightforward algebra, this yields an identical condition to equations 3.2.10 and 3.2.11. This approach is not useful for the parasite invasion conditions, as individuals enter the system through more than one class (see Hurford et al., 2010).

From the initial analysis it appears that parasites infect more vulnerable hosts, but also aim to be generalists. Hosts trade-off between the pressures exerted by different parasite species, aiming to lower this as much as possible, but similarly aim to generalize their defence. This is all as expected, and demonstrates the biological validity of our trade-off.

Simplifications of the above scenario demonstrate that the coevolution of all four species is vitally important, as expected. If one or more species is missing from the system then the trade-off ensures that the dynamics are trivial, with those that have two antagonists splitting their resources as before, while those that have only one concentrate their resources on that one. This demonstrates the importance of such a system, incorporating both multiple hosts and parasites, unlike many previous approaches taken when modelling host-parasite systems.

3.2.3 Evolutionarily singular strategies

The conditions on the invasion fitness for the parasite and host mutants, from generalisations of equations 3.2.6 and 3.2.10 respectively, can be used to determine a great deal about the coevolution of all four species (see Metz et al., 1996). Evolutionarily singular strategies (ESSs) for the system can be calculated for both the parasite and the host by solving for $\frac{\partial u_j}{\partial a'_{1j}} = 0$ and $\frac{\partial w_i}{\partial c'_{i1}} = 0$ at $a'_{ij} = a_{ij}$ and $c'_{ij} = c_{ij}$ respectively. Hence, the ESSs $(\bar{a}_{ij}, \bar{c}_{ij})$ for parasite and host species 1 are found as

$$\bar{a}_{11} = \frac{\beta_1^{21} I_{11}(\beta_1^{12} I_{21} + \beta_1^{11} I_{11})}{2\beta_1^{12}\beta_1^{21} I_{21} I_{11} - 4\beta_1^{11}\beta_1^{22} I_{21} I_{11}^2 + \beta_1^{12}\beta_1^{22} I_{21}^2}, \tag{3.2.17}$$

for the parasite, and for the host as solutions to

$$\begin{aligned} &\bar{c}_{11}(1-\bar{c}_{11}) S_1^* \Big(\bar{a}_{11}(\beta_1^{11} I_{11}^* + \beta_1^{12} I_{21}^*) - \bar{a}_{12}(\beta_2^{11} I_{12}^* + \beta_2^{12} I_{22}^*) \Big) \\ &+ \alpha_1 \big(\bar{c}_{11} I_{12}^* - (1-\bar{c}_{11}) I_{11}^* \big) = 0. \end{aligned}$$

Solving these simultaneously proves difficult, as there are no explicit equations for the equilibria, which depend on the trait values. In this way, the ESSs shift depending on the initial values taken for traits, although it is possible to use these to confirm the location of an ESS. It is also useful to note that there are two ESSs for the host, although one of these may be out of the range of acceptable trait values (i.e. less than zero or greater than 1).

The Jacobian of the canonical equation is described by:

$$J = \begin{pmatrix} \frac{\partial^2 w_1}{\partial c_{11}'^2} + \frac{\partial^2 w_1}{\partial c_{11}' \partial c_{11}} & \frac{\partial^2 w_1}{\partial c_{11}' \partial c_{21}} & \frac{\partial^2 w_1}{\partial c_{11}' \partial a_{11}} & \frac{\partial^2 w_1}{\partial c_{11}' \partial a_{12}} \\ \frac{\partial^2 w_2}{\partial c_{21}' \partial c_{11}} & \frac{\partial^2 w_2}{\partial c_{21}'^2} + \frac{\partial w_2}{\partial c_{21}' \partial c_{21}} & \frac{\partial^2 w_2}{\partial c_{21}' \partial a_{11}} & \frac{\partial^2 w_2}{\partial c_{21}' \partial a_{12}} \\ \frac{\partial^2 u_1}{\partial a_{11}' \partial c_{11}} & \frac{\partial^2 u_1}{\partial a_{11}' \partial c_{21}} & \frac{\partial^2 u_1}{\partial a_{11}'^2} + \frac{\partial^2 u_1}{\partial a_{11}' \partial a_{11}} & \frac{\partial^2 u_1}{\partial a_{11}' \partial a_{21}} \\ \frac{\partial^2 u_2}{\partial a_{12}' \partial c_{11}} & \frac{\partial^2 u_2}{\partial a_{12}' \partial c_{21}} & \frac{\partial^2 u_2}{\partial a_{12}' \partial a_{11}} & \frac{\partial^2 u_2}{\partial a_{12}'^2} + \frac{\partial^2 u_2}{\partial a_{12}' \partial a_{12}} \end{pmatrix}$$

A great deal of information about an ESS can be derived from this matrix. There are three primary properties of concern here. These are the evolutionary stability, convergence stability and mutual invasibility of an ESS.

The first of these, evolutionary stability (ES), determines whether a local mutant may invade a resident population at this point. For this to be locally evolutionarily stable, we require that for parasite species j, $\frac{\partial^2 u_j}{\partial a_{1j}'^2} < 0$ at that point, and similarly for a host species (Geritz et al., 1998). Hence the singularity for parasite species 1 is evolutionarily stable if $\beta_1^{12}\beta_1^{21} < \beta_1^{11}\beta_1^{22}$ (see equation 3.2.6); i.e. intra-species transmission must be higher than inter-species transmission. For host species i, $\frac{\partial^2 wi}{\partial c_{i1}'^2} = 0$, so it is not possible to comment on the evolutionary stability of a singularity.

The convergence stability (CS) of an ESS determines whether that point is an attractor or a repellor, i.e. whether mutation occurs towards or away from the ESS (Diekmann, 2004). This is more complex to calculate in a multi-species case than the evolutionary stability, which generalises directly from a single-species case. Isoclinic stability, or the convergence stability of a singularity for a species in a static environment (when the remaining species are not evolving), occurs if the diagonals of the Jacobian are negative (Kisdi, 2006). However, this is not sufficient for convergence stability in the case where all species are coevolving, and so absolute convergence is introduced. In this, the most extreme path away from the ESS is sought. This is the trajectory which carries the system as far away from the ESS as possible for a given size of mutation to the trait value (Kisdi, 2006). If this path converges to the ESS, then so must all paths, and hence the singularity is convergent stable (Kisdi, 2006). To satisfy this, stronger conditions than necessary are imposed here to guarantee negative eigenvalues; isoclinic stability must hold, and we require that $det(J) > 0$ (Best et al., 2009).

Outcome	ES	CS	IP	MI
No ESS	✗	✗	✗	✗
No ESS, branching unlikely	✗	✗	✗	✓
No ESS, branching unlikely	✗	✗	✓	✓
Branching	✗	✓	✓	✓
CSS	✓	✓	✓	✓
CSS	✓	✓	✓	✗
CSS	✓	✓	✗	✗
Garden of Eden	✓	✗	✗	✗

Table 3.1: The nature of an evolutionary singular strategy given the properties it possesses (Dieckmann, 2002). The properties of evolutionary stability (ES), convergence stability (CS), invasion potential (IP) and mutual invasibility (MI) are used to determine the presence or absence of an evolutionary singular strategy (ESS), whether it is an evolutionary endpoint (a continuously stable singular strategy, or CSS) and whether branching may occur there.

Finally, the mutual invasibility (MI) of a singularity concerns dimorphism, and the ability of a strategy near the ESS to recover when rare, and hence avoid extinction (Metz et al., 1996).This depends on the second term in each diagonal, which must be negative for mutual invasibility to occur (Kisdi, 2006). A final property not available from the Jacobian, that of invasion potential (IP), or attainability, states that a resident population near to the ESS may always be invaded by a mutant with the ESS trait value, and depends upon $\frac{d^2u_j}{da_{1j}^2}$ and $\frac{d^2w_i}{dc_{i1}^2}$ for parasite and host species respectively (Dieckmann, 2002).

These conditions are used not only to determine the nature of the ESS, but also to predict whether evolutionary branching is possible. This is most likely to occur when an ESS is an unstable attractor, near which dimorphism is possible (Geritz et al., 1998). For further information, table 3.1 demonstrates the possible scenarios that could be witnessed. Some important features are the possibility of branching, where selection becomes disruptive and increasing genetic variation occurs, the presence of a continuously stable strategy (CSS), or evolutionary endpoint, a "Garden of Eden" stable state, which the mutant evolves away from, and a scenario in which no stable ESS exists (Rueffler et al., 2006).

It is not possible for us to investigate the Jacobian analytically, as it is not possible to obtain the population equilibrium values analytically. The implicit function theorem is then required when differentiating with respect

to resident traits, as the equilibrium values depend on these. It is therefore necessary for us to investigate this matrix numerically. Similarly, it is not possible to find the isoclines, where the local fitness gradient is zero, as they depend on the equilibrium values, which in turn depend on the trait values (Geritz et al., 1998).

3.3 Results

Simulation of the results follows the method of Dieckmann and Law (1996), concerning the frequency and impact of selection, where evolutionary dynamics occur at a much slower rate than population dynamics (see Drossel and McKane, 2005). This relies on the derivative with respect to the mutant trait-value of the growth rate of the mutant in a population of residents. As a result of the appropriate eigenvalue equations, discussed above, and noting that λ must be small, the dominant eigenvalues may be approximated up to some positive multiplicative coefficients by equations 3.2.6 and 3.2.10 for parasite species 1 and host species 1 respectively. We therefore use the following general equation for species k with trait-value s_k and the appropriate approximation for the eigenvalue E_k:

$$\frac{d}{dt}s_k = f_k(s) \cdot \frac{\partial}{\partial s'_k} E_k(s'_k, s)\Big|_{s'_k = s_k}, \tag{3.3.18}$$

where f_k is the evolutionary rate coefficient (see Dieckmann and Law, 1996; this is initially assumed to be equal for all species, although the effects of varying it are discussed later). This differential equation is calculated for an environment determined by resident trait-values s for all species.

Changes to trait values with time are then investigated, where species can coevolve. It is presumed that $\phi_i = 1$ for hosts, so the trade-off shape is linear, and initial trait values are at the neutral point (0.5 here, where a species is assumed to be a perfect generalist), in order to attempt to separate the effects of hosts and parasites. There are a number of possible cases for different θ values; trade-off curves of both parasite species may be either concave or convex ($\theta < 1$ or $\theta > 1$ respectively).

A look at the pairwise invasion plots for cases with different trade-off strengths demonstrates the outcomes that we expect (see figure 3-2). Pairwise invasion plots (PIPs) indicate when a mutant can invade (shaded) or

(a) (b) (c)

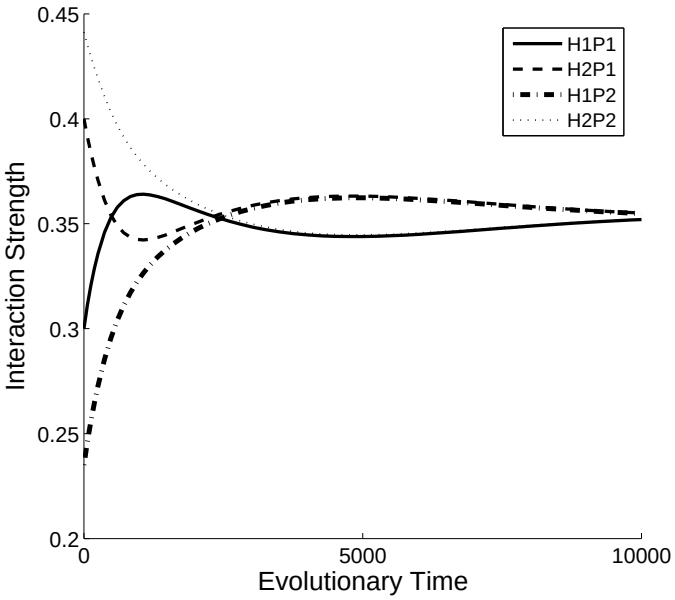
Figure 3-2: Pairwise invasion plots for (a,b) parasite 1 and (c) host 1 in a symmetric environment, with intra-species transmission higher than inter-species transmission. Mutant trait values are on the y-axes, while resident trait values are on the x-axes. Trade-off shapes are (a) strong for the parasites and (b) weak for the parasites, while linear for the hosts, and (c) either strong or weak for the hosts while linear for the parasites.

not (white) depending on its trait value relative to the resident trait value. This property depends on the mutant's invasion fitness at low frequency in a resident population. The population evolves as small mutations occur which move the population off the diagonal (Geritz et al., 1998). If the mutation is successful (i.e. the mutant is in a shaded area) then the mutant population grows and it displaces the resident, to become the new resident (Geritz et al., 1998). In figure 3-2(a) here, for example, this occurs until the population's trait value reaches an extreme, depending on which side of the central point the resident trait value begins. Species with a strong trade-off are likely to evolve to be specialists (figure 3-2(a)), while those with a weak trade-off are expected to evolve towards generalism (figure 3-2(b)).

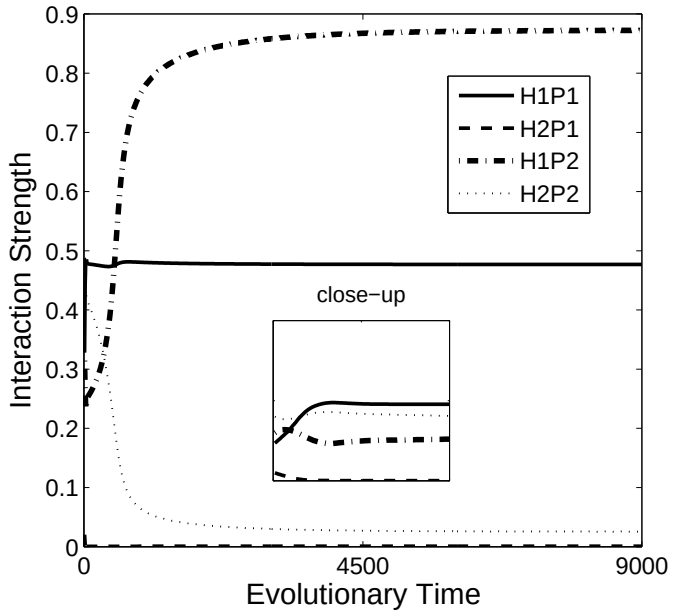
Figure 3-2 demonstrates the evolution of one species only, in a static environment where no other species evolves. In a full analysis this will not be the case. This motivates us to follow the coevolution of trait values for all four species simultaneously. Results are demonstrated in figures 3-3 and 3-4.

In figures 3-3 and 3-4, although mutation rates are taken to be identical for hosts and parasites, the growth rate of mutant hosts in a resident environment is significantly slower than that of parasites. We measure the interaction strength here as a combination of the trait values for both species interacting.

Again we assume a linear trade-off for hosts, which have initial trait values at their respective neutral points. If trade-offs are taken to be linear for parasites as well, then stable limit cycles occur (although see Nuismer et al., 2007 for suggestions that, in reality, these only occur if the strength of co-

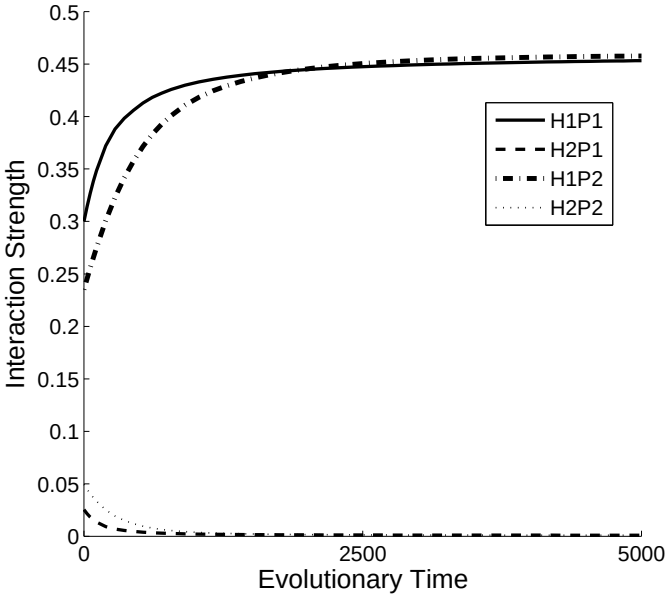


(a) Weak trade-off for parasites ($\theta_j = 2$)

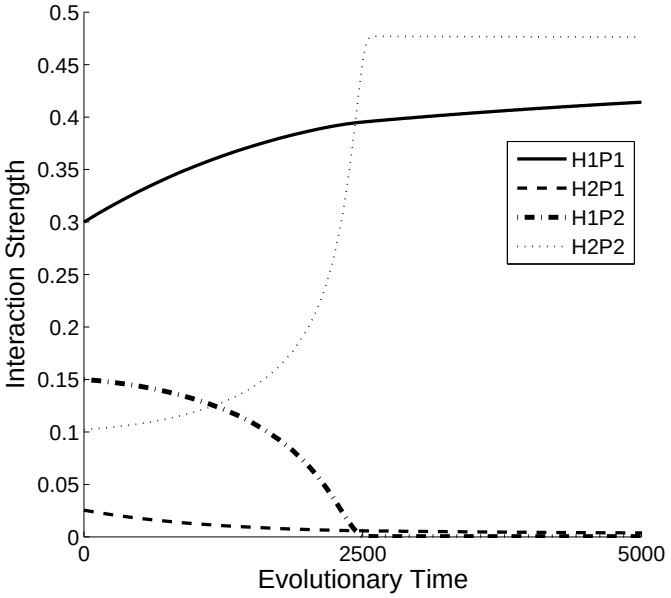


(b) Mixed trade-offs for parasites ($\theta_1 = 2, \theta_2 = 0.5$)

Figure 3-3: Examples of the evolution of interaction strengths (as a result of species trait values) with time in a symmetric environment, with intra-species transmission higher than inter-species transmission. Rates of change of trait values are calculated directly from the growth rates of mutant traits in a resident population. Trade-off shapes are linear for hosts and (a) weak or (b) mixed for parasites. The inset in (b) shows the initial dynamics of the system before the slower host mutations have had an effect. Note that H_1P_1 and H_2P_1 represent interactions between parasite species 1 and host species 1 and 2 respectively, while H_1P_2 and H_2P_2 represent the same for parasite species 2.



(a) Parasites in the same host



(b) Parasites in opposite hosts

Figure 3-4: Examples of the evolution of interaction strengths with time in a symmetric environment, where the trade-off shapes for both parasites are strong ($\theta_j = 0.5$). Different endpoints occur as a result of the initial equilibrium values for susceptible and infected hosts. These result in parasites in (a) the same or (b) opposite hosts.

evolutionary selection exceeds a certain threshold). In figure 3-3(a), where $\theta_1, \theta_2 > 1$, generalist parasites evolve. Here the trait values of parasites can be seen to evolve towards their neutral points. Note that the system cycles around the neutral point until both parasites are perfect generalists. This corresponds to figure 1-1(d).

For the case where the trade-offs for the parasites are strong for one parasite and weak for the other (figure 3-3(b)), we obtain the coexistence of a relative generalist and an extreme specialist in the same hosts. Note that this system takes much longer over evolutionary time to equilibrate than other cases, due to the slow growth rate of mutant host populations. Hence we see different dynamics over short (figure 3-3(b) inset) and long (figure 3-3(b)) time-scales. Before the hosts are able to react to the presence of the parasites, we see a generalist host that is more focused on the opposite host to the specialist (interactions H_1P_1 and H_2P_2 are stronger than H_2P_1 and H_1P_2 respectively), whereas once the host mutant populations have had an effect, we see both the relative generalist and the specialist parasite are more focused on the same host (interactions H_1P_1 and H_1P_2 are stronger). As a host which contains only a generalist would be able to focus its defensive efforts on that parasite, and hence, as can be seen from equation 3.2.9, the parasite would not target it, we see only comparative generalists here. This system demonstrates specialization asymmetry, and indeed nestedness (as far as that is plausible in such a small system) over longer evolutionary time-scales, corresponding to figure 1-1(a).

For the case where both $\theta_j < 1$ (figure 3-4), specialists always evolve. These can evolve to be in the same hosts (i.e. H_1P_1 and H_1P_2 evolve to the same extreme, figure 3-4(a)), or in opposite hosts (H_1P_1 and H_1P_2 at the opposite extremes, figure 3-4(b)). These scenarios bear resemblances to specialization asymmetry (figure 1-1(b)) and compartmentalization (figure 1-1(c) where the network is split into separate sub-networks that are not linked to one another) respectively.

3.3.1 Initial trait values

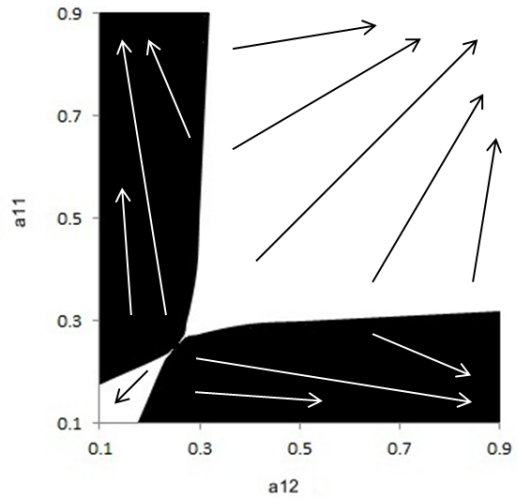
From the parasite conditions for invasion, it can be seen that the behaviour of the cases depends heavily on the initial equilibrium conditions, which are a result of the initial trait values. The behaviour of each scenario pivots

around which side of case-specific points the initial trait values lie, similarly to which side of an evolutionarily stable strategy an initial point lies in a pairwise invasion plot (see figure 3-2). Each case has only a limited number of evolutionary end points for trait values, and evolves to these. An analysis of the initial trait values shows interesting results.

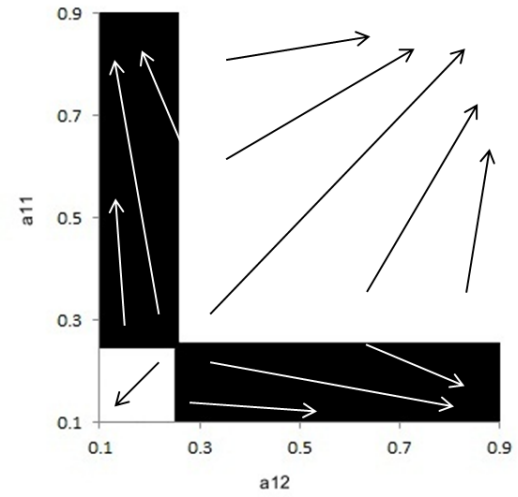
For the case of a weak trade-off for both parasites (figure 3-3(a)), although generalists occur almost exclusively in "opposite" hosts, the interaction strengths evolve to be so similar that only a very careful inspection can detect the difference. In other words, two generalists occur, each slightly more dependent on a different host. If one parasite has a strong trade-off and the second a weak trade-off, a relative specialist and a relative generalist occur respectively, in the same host (figure 3-3(b)). This demonstrates specialization asymmetry, and occurs for all initial trait values. For the case where both parasites demonstrate a strong trade-off (figure 3-4), figure 3-5(a) shows the initial trait values which lead to specialization asymmetry.

Increasing the hosts mutation rates (f_k from equation 3.3.18) serves to alter the shape of the curves towards that found in figure 3-5(b). The much faster generation time of parasites has been used in the past to justify the study of their evolution alone, as opposed to a full coevolutionary system (Vázquez and Aizen, 2004). Even including coevolution, however, it has been shown by Best et al. (2009) that different mutation rates do have a significant effect. In our simulations, where the growth rates of mutant host populations in resident environments appear significantly slower than those of mutant parasite populations, increasing the mutation rates of hosts in comparison to parasites greatly increases the chances of specialization asymmetry occurring for a wider range of initial trait-values when a strong trade-off exists for parasites. This indicates not only the circumstances under which we might expect to see asymmetry, but also a possible line of experimentation to take in order to validate our results.

Phase diagrams of all possible outcomes across different parasite and host trade-off strengths are given in figure 3-6. From this, we see that varying the host trade-off strength yields either limit cycles or specialization asymmetry. The linearity of parasite trade-offs appears to be sufficient to ensure that the system remains in a limit cycle, and does not equilibrate (as seen in figure 3-6(a) when parasite trade-off strengths are linear), whenever host trade-offs are either both strong or both weak. This suggests that the parasite trade-offs



(a) Low host mutation rate



(b) High host mutation rate

Figure 3-5: A sketch of the end-points of evolution, in terms of species-richness, for different initial trait values for a strong trade-off ($\theta_j = 0.5$) for parasite species, where host initial traits are at the neutral point. Black areas denote those initial trait values that evolve to have parasites in opposite hosts, while white areas denote those for which parasites evolve to be in the same host for (a) a low host mutation rate and (b) a high mutation rate for hosts. Arrows indicate the direction of evolution of interaction strengths. Note that the trait value for each parasite with respect to the second host is determined by the trait value for the first, and hence is not included in these plots.

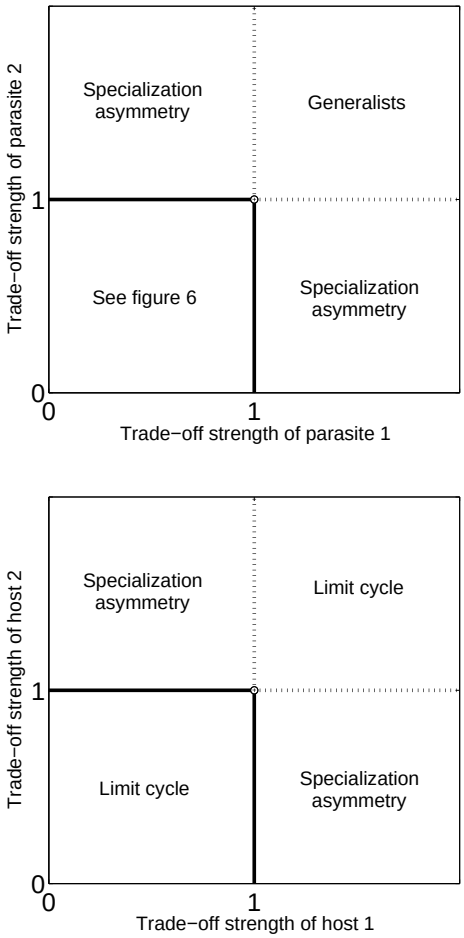


Figure 3-6: Phase diagram of equilibria of a system as (a) parasite and (b) host trade-off strengths are varied (remaining trade-off strengths are linear). Dashed lines represent limit cycles, and solid lines specialization asymmetry.

are often what stabilize the system, possibly due to the relative growth rate of parasite mutant populations. We also see that generalist parasites do not appear to exist when their trade-offs are linear, even when trade-offs of hosts vary. An example of a limit cycle is given in figure 3-7.

The addition of both recovery terms and coinfected classes to the system, computed numerically, demonstrated increased likelihoods of specialization asymmetry occurring, again dependent on both the initial trait-values and relative mutation rates of species.

Allowing only parasites or hosts to mutate demonstrates the importance of the coevolution of all four species. If parasites only are allowed to evolve, then we obtain similar results to figure 3-6(a) with low host mutation rates, except that we see specialization symmetry, as opposed to asymmetry, occurring

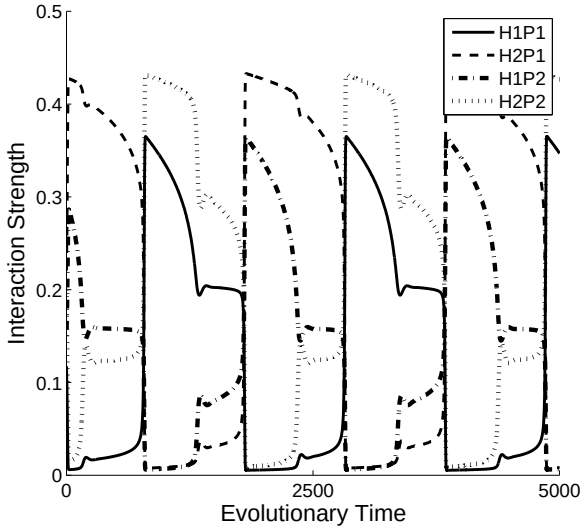


Figure 3-7: Example of the evolution of interaction strengths with time in a symmetric environment, where the trade-off shapes for both hosts are weak ($\phi_i = 2$), and stable limit cycles are seen.

when parasite trade-offs are mixed. Hence, parasites are found primarily in opposite species, reducing the likelihood of this scenario displaying nestedness at a larger dimension. We note that the general shape of the pairwise invasion plots in figure 3-2 will be unaltered for each parasite by differences in the other parasite's trait values, and, as the hosts are unable to evolve, we can determine the nature of evolution immediately for each parasite species. If a parasite species has a weak trade-off, then it will approach a continuously stable strategy, and if it has a strong trade-off, then the evolutionary repellor will ensure that it evolves to an extreme, and branching is unlikely.

In comparison, allowing hosts only to evolve, perfect generalisms are exclusively formed. A comparison of the pairwise invasion plots shows that these have evolved to continuously stable strategies whether the trade-off strength is strong or weak. We see, therefore, that the coevolution of all four species is vital, in allowing for the presence of both specialization asymmetry and limit cycles. Without this, we rarely see evidence for anything approaching nestedness in our system.

3.3.2 Abundance

In our toy model we included both abundance and phenotypic matching as motivators for nestedness. In order to justify the claim that the model is

compatible with the concept of abundance as a driving force (see Graham et al., 2009; Poulin and Morand, 2004; Vázquez et al., 2005, 2009a), we investigate here the manner in which hosts of different abundance influence the model outcomes. This is especially important over evolutionary, as opposed to ecological, timescales.

There are two aspects of abundance to be compared to the model presented here. Firstly, does increased abundance of a species indeed lead to a higher number of links occurring, and secondly, does asymmetry in the assignment of links lead to nestedness? The first of these is compared to the model, while the second is a question that remains independent of the model.

We note that, if nestedness is a result of abundance, then we would expect it to occur in mutualistic and predator-prey webs, as the number of links of a species is associated solely with its abundance and not with the nature of those links. This is indeed evident (see Montoya et al., 2006), particularly in mutualistic networks, which display more nestedness than would be expected from a random bipartite network (Bascompte et al., 2003). However, if this is the case then why would certain web-types display more nestedness than others (see Hernandez and Sukhdeo, 2008; Lafferty et al., 2006b)? This promotes the thinking that abundance alone cannot explain patterns of nestedness in ecological networks. A further question concerns whether or not abundant species are more likely to have links simply as they are more likely to interact with other species due to their abundance, or whether this is due to the inherent benefits of interacting with a more abundant species. Our model attempts to address this, proposing that there is an evolutionary advantage to interacting with a more abundant species.

For our trade-off model, note in equations 3.2.6 that the invasion potential of a parasite depends on the relative abundances of the different host species. The terms I_{11} and I_{21} are the combination of infection prevalence and total population size, and hence reflect that a larger population will increase invasion potential. This was also computed numerically, where it was observed that, in a symmetric environment, increasing a species' population size could drive a parasite to preferentially interact with that species (see figure 3-8). Here we look at parasite 1 interacting with host 1 and see that, although this is still dependent on initial values for traits, increasing the abundance of the host species clearly increases the likelihood of the parasite evolving to interact with that species.

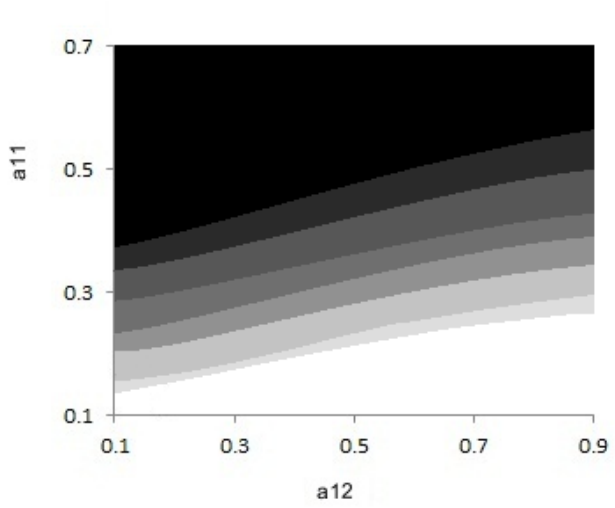


Figure 3-8: Regions of initial trait values for both parasites which, under coevolution with a strong trade-off, lead to parasite 1 occurring in host 1. Filled areas denote regions of initial trait values which see parasite 1 evolving to be in host 1, and successively lighter shades denoting the increase in size of the region as the relative abundance of host species 1 increases.

3.4 Discussion

This model provides evidence for both specialization symmetry and asymmetry, but it is difficult to comment on nestedness from this. A similar model might be sufficient for larger networks, where a species-poor host could still contain more than one parasite, allowing parasites to split a host's defence. Additionally, if hosts were to trade defensive properties in an alternative manner (for example through reduced birth-rate, or the inability to reduce transmission completely to zero) this could allow generalists to exist alone in a species-poor host.

There is a great deal of mixed evidence, from a number of different ecosystems under varying conditions, for nestedness in host-parasite interactions (Graham et al., 2009). In those systems in which nestedness occurs, generalists will be in comparatively species-poor hosts, as they may occur in both species-poor and species-rich hosts. In this sense the model can be related to nestedness, although its size makes a full comparison unreliable. The model also supports the idea that a strong trade-off leads to specialists, while a weak trade-off promotes generalism, in accordance with the Levins fitness set approach (Levins 1962, cited in Rueffler et al., 2006).

From the results obtained here it is evident that initial trait values are

very important to the final equilibrium of a population. This may be useful in predicting responses when a species is added to or removed from a system, as such an event will be followed by evolution of the system in a direction dictated, to a certain extent, by the equilibrium values of the system prior to the alteration. We have to be wary, however. Even here, due to the large number of parameters assumed and the stochasticity of interactions, when our models are based on observed phenomena we are mainly able to reproduce natural phenomena, and will likely have much less success predicting them, especially as we do not include speciation and invasions (Drossel and McKane, 2005; Gaedke, 1995).

In a similar multi-species host-parasite model, Bennett and Bowers (2008) investigated the basic reproductive (stressing the role of parasite species) and depression (stressing the role of host species) ratios, calculating the invasion conditions for additional strains to enter a system, and the existence, feasibility and stability of equilibria in the system. Our model extends their idea to investigate the evolution of interaction strengths, and the network structure that such systems take. In addition, we include a density-dependent natural death rate (as opposed to density-dependent birth), which has been shown to be important for parasite branching (Best et al., 2009; Pugliese, 2002). Despite this, we observed no branching in our system, although higher dimensional systems may branch (see Chapter 4).

One crucial factor concerning this model is that it is dynamical. A structural property can be described and predicted by this dynamical model, explaining an aspect of static models that cannot be explained through a simple trophic hierarchy. In a similar manner, Cattin et al. (2004) discovered that diet range is a result of phylogenetic constraints and adaption, which mirrors our findings here; that species interactions could be a result of the adaption of phenotypes. This helps in clarifying how parasitic associations may be motivated, and, to a certain extent, investigates the effects which parasite in a host species have on one another. This toy model can now be expanded to include a more realistic system containing many more species (see Chapter 4). Analysis of a larger system will then enable questions on the nestedness of the system as a result of trade-offs to be answered. However, our model only allows for investigation into certain aspects of host-parasite interactions, yet other coevolutionary model types also suggest different causes that might lead to aspects such as trait cycling and the occurrence of gen-

eralist parasites, including polymorphism of resistance genes and high costs to virulence (Sasaki, 2000). Ings et al. (2009) advocated the construction of a new, individual-based perspective, incorporating foraging theory and metabolic theory of ecology. Here we have proposed an evolutionary equivalent.

The primary purpose of this chapter was to lay the foundation for a model which could explain patterns of nestedness in ecological networks. In order to do so, this model needs to be repeated at a larger scale for a host-parasite network. Note, however, that Bennett and Bowers (2008) have shown that, for such systems to reach equilibrium, there must be an equal number of both host and parasite strains or species. Although the essential nature of the model need only be extended to a larger dimension, the manner in which traits trade off over higher dimensions does raise potential problems. In order to calculate trade-offs, ecological and evolutionary time may be separated, and only two traits be allowed to mutate, with respect to one another, in each evolutionary time step (see Chapter 4). In addition, host-parasite coevolution studies using gene-for-gene models have shown that increased numbers of loci can lead to avirulence (Sasaki, 2000) or destabilization (Kopp and Gavrilets, 2006), an aspect which is worth considering here when the number of traits for a species increases. Given the evidence for nestedness in other systems (Bascompte et al., 2003; Vázquez and Aizen, 2003, 2004), the adaption of this model to these systems, particularly mutualistic networks, could also be used to corroborate any conclusions reached (see Chapter 5).

Many other factors are considered as possible motivators for the species-richness of parasites in hosts (see Feliu et al., 1997; Marcogliese, 2002; Montoya et al., 2006; Morand and Poulin, 1998; Nunn et al., 2003; Poulin and Leung, 2011; Poulin and Morand, 2004), which focus on the characteristics of the hosts. It is becoming increasingly clear that the interests of the parasites are also important factors in this, and, in fact, both motivating factors are likely to be of importance. There have been very few models which investigate the coevolution of a host-parasite system using adaptive dynamics (but see Best et al., 2009, 2010; Caval and Ferriere, 2010), and these focus primarily on the discovery of a coevolutionary stable strategy. Other methods, such as multi-locus genetics, could be used to extend and complement our results (see Kopp and Gavrilets, 2006). In that sense, every additional approach to or analysis of a coevolutionary system adds to the field of coevolutionary

ecology.

With the aim of coevolution in mind, it has been observed that, according to game theory, predators may be responsible for the presence of additional prey species through induced branching (McGill and Brown, 2007). Is it possible that parasites have such an effect? This could presumably only happen if parasites exerted similar levels of pressure to predators, which is unlikely (Poulin, 2010). Branching in our model could, however, potentially explain nestedness, as parasites would be found in similar hosts. This would lead to results similar to figure 3-4(a), an aspect which could be investigated further.

A further step from here is to investigate the effect that the position of a host in the network as a whole has on its parasites, and how this fits in with the observations made here. The position of a host species in the network is considered a potential driving factor in determining its parasitic composition (Chen et al., 2008; Vázquez et al., 2005). This has been looked at to a greater extent than parasite interactions with each other in the past (Cobey et al., 2010), but open questions still remain, specifically with larger networks that include both multiple hosts and multiple parasites together. We address one of these questions in Chapter 6.

3.5 Conclusion

The results of the model indicate that the hypothesis of resource trade-off driving a link between specificity and species richness appears to be plausible. It can certainly be used to model interactions between hosts and parasites, which should yield interesting results when used on a larger scale. This also highlights the importance of factors such as host mutation rates in coevolutionary systems, even when these rates are low.

Using such information as our results for the mutation rates and initial trait values, our model helps to predict the circumstances under which we might expect patterns such as specialization asymmetry to occur. We would predict the presence of specialist parasites in species-rich hosts to be more likely if the hosts had higher mutation rates, and in systems in which parasites are more closely related, are more likely to originate in similar hosts or appear as generalists. Given the relationship between specialization asymmetry and nestedness, we would therefore expect nestedness under similar

circumstances, and anti-nestedness the remainder of the time.

This model demonstrates that dynamic coevolution of the network is vitally important in accounting for parasites, as it demonstrates how the dynamics could influence structural properties. In particular, it demonstrates the importance of the coevolution of both hosts and parasites in such a scenario. Parasites, therefore, are not a characteristic to simply be transposed onto a system with no regard to their effects on one-another. Much like interactions in conventional food webs, the influences of different parasites can alter the entire structure of a host-parasite network.

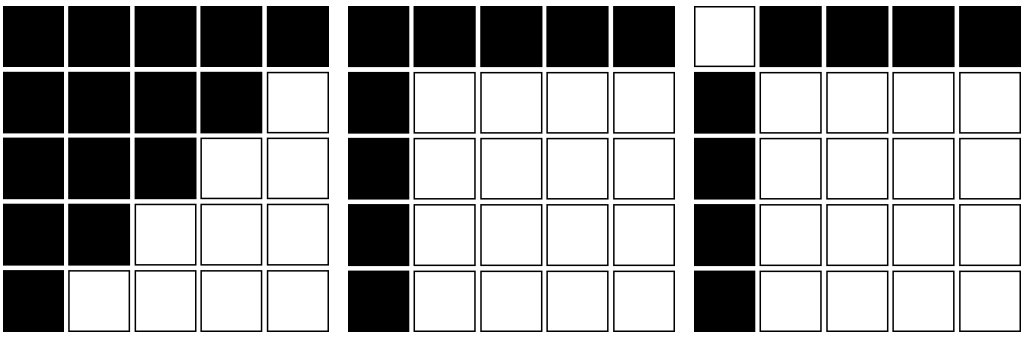
CHAPTER 4

Higher Dimension Host-Parasite System

In this chapter, we discuss in greater depth some of the motivation for our model in Chapter 3, and then expand it to a higher dimension and discuss the results obtained. To reiterate the definitions we have used previously, a host-parasite interaction matrix is defined to be perfectly nested when the parasite species compositions in hosts containing few species are subsets of the parasite species compositions in hosts containing many species (Patterson and Atmar, 1986; Ulrich et al., 2009). A similar pattern, called specialization asymmetry, is present if specialist hosts interact with generalist parasites, and specialist parasites interact with generalist hosts (Corso et al., 2008). This will lead to nestedness if generalist parasites and hosts also interact with one another (see figure 4-1). There has been evidence for both patterns in host-parasite networks (Poulin, 1997; Vázquez et al., 2005).

4.1 Nestedness

Nestedness has come to be regarded as an increasingly important concept in the study of ecological systems, and is one of the most studied aspects of bipartite networks (Corso et al., 2008), yet it is fraught with controversy (Almeida-Neto and Ulrich, 2011; Corso et al., 2008; Greve and Chown, 2006; Podani and Schmera, 2012; Rodríguez-Gironés and Santamaría, 2006). Perfect nestedness is, verbally, a well-defined and understood concept, as demonstrated



(a) Perfect nestedness (b) Minimum fill (c) Specialization asymmetry

Figure 4-1: Examples of nestedness and specialization asymmetry. In this case, columns represent one type of species (for example, host species) while rows represent another (for example, parasite species). A filled square represents interactions between the appropriate species, and an empty square indicates that no interactions occur. The first two of these examples display both properties, where (a) demonstrates perfect nestedness and specialization asymmetry, while (b) demonstrates the same at minimum fill. The final example (c) shows specialization asymmetry but not nestedness (according to whether or not the definition of nestedness requires species-poor assemblages to be subsets or proper subsets of species-rich assemblages).

above. Mathematically, however, it has proved difficult to measure and compare (see Almeida-Neto et al., 2008; Almeida-Neto and Ulrich, 2011; Araujo et al., 2010a; Atmar and Patterson, 1993; Brualdi and Sanderson, 1999; Corso et al., 2008; Cutler, 1991; Galeano et al., 2009; Podani and Schmera, 2011; Wright and Reeves, 1992). A large number of different metrics have been devised that aim to measure nestedness, and there is no over-riding best-choice (Almeida-Neto et al., 2008). Metrics measure nestedness in a number of ways, counting unexpected species presences, or species absences, or the number of supersets, amongst others (Ulrich et al., 2009). Hence a variety of metrics is used here, each of which measures nestedness, and therefore anti-nestedness (see section 4.5), in a different manner. A thorough review of the topic is available in Ulrich et al. (2009), with additional comments in Ulrich and Gotelli (2007) and Podani and Schmera (2012).

4.1.1 Measures

Before we describe our model and discus our motivation, we wish to briefly discuss the metrics used to test for nestedness in our bipartite network, where we are no longer able to simply base our measures of nestedness on the four

options available in a 2x2 system. Originally, when nestedness was first defined, it was investigated in species-site matrices, where different assemblages of species were present in different habitat fragments, such as islands or mountain tops (Patterson and Atmar, 1986). The concept has since expanded to include interaction matrices. In these matrices, species fit into one of two classes; for example host or parasite species, or plant or herbivore species. Atmar and Patterson (1993) proposed one of the earliest and most popular metrics for measuring the nestedness of a system, which measures the system's thermodynamic disorder. This metric measures not the nestedness of a system explicitly, but instead stochasticity in the system. It accomplishes this by looking for unexpected presences and absences in the matrix of population and habitat co-occurrences (Atmar and Patterson, 1993). Based on this concept, the nestedness temperature calculator (NTC) was created to calculate the temperature of a network (Atmar and Patterson, 1995).

The temperature metric was aimed at detecting patterns of nestedness created by species extinction orders on islands or fragmented habitats (Patterson and Atmar, 1986). The extinction order was expected to remain set in different habitats, so that species would always go extinct in a particular sequence. This would then lead to nestedness and robustness (Burgos et al., 2007). An important aspect of this is that it was assumed that evolutionary equilibrium was never reached; we were only ever to see snapshots in time of the species composition (Atmar and Patterson, 1993).

A number of metrics have been designed based on the NTC (see, for example, Greve and Chown, 2006). Guimarães Jr and Guimarães (2006) developed ANINHADO, a program used to perform fast, automatic temperature calculations for many matrices, using many null models for comparisons. Another metric, BINMATNEST, was also developed to improve on aspects of the NTC, including the packing, uniqueness of the line of perfect order and the creation of null models, allowing for comparisons among data sets (Rodríguez-Gironés and Santamaría, 2006).

The above extensions are all essentially based on the temperature metric, and are measured from an isocline of perfect nestedness created for each matrix with its specific fill. This means, then, that the above metrics are rather measures of symmetry around a perfect nested line (Almeida-Neto and Ulrich, 2011) than direct measures of disorder. Other methods have been suggested that take measurements from the packing corner (generally

the top left) as opposed to this isocline, which claim to be more accurate (Corso et al., 2008).

In fact, there have been a plethora of more recent, alternative, metrics devised to measure nestedness. These include those of Cutler (1991), which counts unexpected presences and absences, the C metric of Wright and Reeves (1992), which investigates the number of times that species presence at a site correctly predicts its presence at a richer site, and the discrepancy metric (BR) of Brualdi and Sanderson (1999). Corso et al. (2008) have also created an estimator based on the Manhattan distance, which has since been expanded on by Araujo et al. (2010a). This focuses again on the sum of distances between occupied elements of the matrix, adjusting for occupancy, and is claimed to be more mathematically robust than previous measures (Araujo et al., 2010a). Podani and Schmera (2011) have also introduced a mathematically rigorous framework, which looks at three separate indices (similarity, relative species replacement and relative richness difference), allowing for networks to lie anywhere between three model extremes; perfect nestedness, anti-nestedness and perfect gradient.

One of the most recent, and seemingly successful, metrics, the nestedness metric based on overlap and decreasing fill (NODF), was created by Almeida-Neto et al. (2008), based on decreasing fill and the paired overlap of matrices. This metric seeks to improve on the overestimations that the matrix temperature and discrepancy metrics made in measuring nestedness. It also deals with the transposition of matrices, which is especially useful in terms of interaction matrices (Almeida-Neto et al., 2008).

In differentiating the metrics, it can be seen that there are three major metric types, those which calculate based on (i) temperature, (ii) the presence of gaps in the matrix or (iii) overlap. The first of these, while a founding method, should probably not be applied to interaction matrices, for which it has been argued that NODF and discrepancy are the most appropriate (Ulrich et al., 2009).

Weighted Metrics

In an additional complication when measuring nestedness, the majority of metrics have been designed for binary presence-absence networks. In the temperature metric of Atmar and Patterson (1993), population size was expected (due to the extinction order assumption) to be ordered such that the

largest populations were present in the top left of the species-site matrix, although there was no evidence for this implication that species would have larger populations on islands containing more species. In reality, this is an important consideration, as interactions which may be of vastly different importance to species are represented as identical in a binary matrix (Ulrich and Gotelli, 2010).

In the interests of correcting this, the metric of Corso et al. (2008) was extended by Galeano et al. (2009) to create a metric for weighted networks, the Weighted-Interaction Nestedness Estimator (WINE). This metric, however, was quickly followed by a weighted version of the NODF metric, WNODF, which improved on the results obtained by WINE (Almeida-Neto and Ulrich, 2011). This new WNODF metric, one of the most recent of all metrics at the time of writing, compares the nestedness of both species composition and incidence. It is more successful than WINE at differentiating between different types of nested networks, and can identify anti-nested or compartmentalized networks with much greater success (Almeida-Neto and Ulrich, 2011). Additionally, this metric can differentiate between the nestedness due to rows and columns separately.

Null Models

When using one of the above metrics to measure nestedness, it is important to remember that, due to differences in matrix size, shape and fill, results between different systems will most likely be incomparable. Hence the use of null models is critical, as it allows for the comparison of different systems (Brualdi and Sanderson, 1999). The Z-score obtained from these is then used to standardize results (Almeida-Neto et al., 2008; Ulrich et al., 2009).

Many different null model types have been suggested. These are based primarily on whether or not row and column totals should be fixed, or if all row and column sums should be equiprobable (Ulrich et al., 2009). This has a huge impact on the likelihood of nestedness occurring, and may in fact alter results completely (Ulrich et al., 2009). Fixing row and column totals for null models based on the data being analysed means that the number of null matrices which can be constructed within these constraints is very limited, making the actual matrix much less outstanding than if an equiprobable null model were used. Research suggests that constrained null models (where row and column totals are fixed from the data) should be used for all except very

full or very empty matrices, as these null models result in fewer type I errors. Unfortunately, this may lead to more type II errors, with the null hypothesis of no nestedness failing to be rejected when it should have been (Ulrich et al., 2009).

NODF, while a successful and efficient metric which yields greatly improved results to, for example, the discrepancy and temperature metrics for the equiprobable null model, actually yields very similar results to other metrics for the fixed-fixed null model (Almeida-Neto et al., 2008). The choice of null model for comparison, therefore, is of great importance.

Criticisms

A number of metrics fall prey to inconsistencies in their measurements (Ulrich and Gotelli, 2007). Metrics should be insensitive to matrix size, shape and fill, as well as transformation and occurrence inversion (the switching of presences and absences, Ulrich et al., 2009), and indeed ideally the packing process. In fact, one of the most successful metrics, NODF, demonstrates increasing nestedness with matrix fill, although the designers claim that this is a property of nestedness, not an artefact of their metric (Almeida-Neto et al., 2008).

There are a number of steps to be taken when measuring nestedness. Firstly the matrix must be packed, then measured, and finally compared to null models (Ulrich et al., 2009). Criticisms abound for the manner in which each metric approaches each step (Ulrich et al., 2009). Rodríguez-Gironés and Santamaría (2006) introduce a metric which is better at packing than Atmar and Patterson (1995), improving on their results. Indeed, Podani and Schmera (2012) argue against the use of models that require packing at all.

The temperature metric has also been shown to have flaws in measuring, for example by incorrectly increasing nestedness with the addition of random singletons (due to the manner in which it standardizes matrices for size not occupancy, see Greve and Chown, 2006). Unfortunately, corrections suggested for this run into problems of their own, with some temperatures outside of the allowed range, and a lack of confidence in their performance (Ulrich et al., 2009). NODF has also been shown to be highly sensitive to small changes in data, making it less suitable for real-world measurements (Podani and Schmera, 2012). Many metrics use an inappropriate null model, giving misleading results (Ulrich and Gotelli, 2007). It is clear that there is not yet a consensus on the best method for measuring nestedness.

One further element of debate between different metrics is the influence on nestedness that different species with an identical number of interactions have (Almeida-Neto and Ulrich, 2011; Araujo et al., 2010a; Podani and Schmera, 2012). The conclusion to this depends on whether the definition for nestedness requires species-poor assemblages to be subsets, or proper subsets, of species-rich assemblages. Although we would advocate the use of subsets (McQuaid and Britton, 2013a; Podani and Schmera, 2012), here we have also used metrics which requires proper subsets, as at the time of simulation the NODF metric appeared to be the most reliable. This could lead to incorrectly accepting a null hypothesis of no nestedness.

We see, therefore, that many different metrics have been suggested to measure the nestedness of a matrix (Araujo et al., 2010a; Corso et al., 2008; Cutler, 1991; Galeano et al., 2009; Podani and Schmera, 2011; Wright and Reeves, 1992). Each has its own particular drawbacks and caveats. This thesis uses the popular temperature index of Atmar and Patterson (1995, 1993), the discrepancy metric (Brualdi and Sanderson, 1999) and the more recent NODF metric (Almeida-Neto et al., 2008), as well as the weighted WNODF metric (Almeida-Neto and Ulrich, 2011). One particular drawback to the method used here is that many sources have found that nestedness in the real world is much less evident in smaller networks, particularly those of 50 or fewer species (Guimarães Jr and Guimarães, 2006; Joppa et al., 2010; Santamaría and Rodríguez-Gironés, 2007). In addition, many of the results quoted above to support the presence of nestedness in networks rely on binary data. While this was considered here, we also investigated a system in its weighted form, where the strength of interactions was taken into account through the population size.

When constructing null models, we chose to re-sample keeping row and column sums fixed, as this should lead to more type II errors and so our results would be less likely to incorrectly find nestedness. We used a larger number of matrices than the default for computing confidence limits, as this was suggested for smaller matrices (Almeida-Neto and Ulrich, 2011). The matrices were packed according to species and weight.

4.1.2 Evidence

A variety of studies across a range of interaction and habitat types claim to have found nestedness (Bascompte et al., 2003; Duponte et al., 2003; Guimarães Jr and Guimarães, 2006; Guimarães Jr et al., 2007; Ollerton et al., 2003; Poulin, 1997; Santamaría and Rodríguez-Gironés, 2007; Vázquez et al., 2005, although see Dormann et al., 2009; Ulrich and Gotelli, 2007; Worthen and Rohde, 1996), from scavenger communities (Selva and Fortuna, 2007) to anemonefish and anemones (Ollerton et al., 2007). The majority of these use either the temperature metric of Atmar and Patterson (1993), or a metric based on the same basic principle. However, more recently both Graham et al. (2009) and Joppa et al. (2010) have found patterns of nestedness in a range of mutualistic, host-parasitoid and host-parasite networks using the NODF metric (Almeida-Neto et al., 2008). These studies were the largest we know of to date performed on interaction matrices, and were careful to bear in mind the caveats of previous works.

The work of Joppa et al. (2010) in particular used both the temperature and NODF metrics, as well as a number of null models for comparison. Using the most appropriate null model, the temperature metric discovered more nested networks than expected by chance alone, while NODF found more nested mutualistic networks and both more nested and anti-nested host-parasitoid networks than expected by chance (Joppa et al., 2010). These results agree with Bascompte and Jordano (2007), stating that, although the addition of parasites should increase nestedness (Lafferty et al., 2006b), we would expect mutualistic networks to be significantly more nested than antagonistic networks, which are often compartmentalized (Lewinsohn et al., 2006). Hence, we hope to find in our model both significantly more significantly nested and significantly more significantly anti-nested host-parasite networks than expected by chance.

4.1.3 Causes

There is much debate on the causes of nestedness in interaction networks, with suggestions of species abundance, phenotypic complementarity and asymmetric interaction strength as the primary potential driving forces (Almeida-Neto and Ulrich, 2011; Bascompte and Jordano, 2007; Ulrich et al., 2009). These can be explained as follows. Firstly, it is assumed that more abundant species

will be more likely to have interactions (passive sampling), which then leads to nestedness (Araujo et al., 2010b; Vázquez et al., 2005). However, some research has called this into question (Corso et al., 2008; Rohde et al., 1998). Specifically, are generalists generalists because of their abundance, or *vice versa*, and does species abundance necessarily lead to the frequency with which that species interacts (Bascompte and Jordano, 2007; Santamaría and Rodríguez-Gironés, 2007; Stang et al., 2007)? Although the frequency with which individuals of a species are observed is likely to be related to their abundance, influencing their empirical "specificity", this does not explain all of the nestedness observed in systems (Krishna et al., 2008; Nielsen and Bascompte, 2007), nor does it explain the presence of significantly anti-nested host-parasite networks (Joppa et al., 2010) and other patterns of nestedness (Hernandez and Sukhdeo, 2008; Lafferty et al., 2006b). Indeed, a number of sources highlight the importance of a variety of simultaneous mechanisms leading to nestedness (Bastolla et al., 2009; Fontaine et al., 2009; Rezende et al., 2007; Vázquez et al., 2009a). Krishna et al. (2008) even demonstrate that, although abundance and trait matching separately can be seen to influence nestedness, considering the two together provides a far better predictor.

Secondly, phenotypic complementarity refers to trait matching between species, for example in nectar holder size and length (Rezende et al., 2007; Vázquez et al., 2009a). An equivalent idea in antagonistic webs to phenotypic trait complementarity in mutualistic webs is the concept of barriers to exploitation, where it is only worthwhile for a species to target another which is not protecting itself (Santamaría and Rodríguez-Gironés, 2007). Both of these concepts appear to be important in explaining interaction patterns, leading to forbidden links and overlapping trait-values (Bascompte and Jordano, 2007; Santamaría and Rodríguez-Gironés, 2007). The asymmetry of interaction strengths refers to the tendency of ecological specialization to create these forbidden interactions, between species which are unable to connect (Jordano et al., 2006; Ulrich et al., 2009). This is similar to phenotypic complementarity in that it refers to a kind of "uncomplementarity," and occurs due to the values of phenotypic traits.

One point of contention in terms of nestedness is the effect of sampling bias. Previously, this was used by Blüthgen et al. (2008) to explain nestedness, throwing the question of the occurrence of extensive patterns of nestedness into doubt. However, this calculation was conducted using the temperature

metric, unlike more recent data (Graham et al., 2009; Joppa et al., 2010), which corrects for matrix dimension and fill. Additionally, some sources (Banăsek-Richter et al., 2004; Gibson et al., 2011; Nielsen and Bascompte, 2007; Vázquez and Aizen, 2006) have shown that sampling effort has very little effect on nestedness, specifically when using the NODF metric rather than temperature (Rivera-Hutinel et al., 2012), and indeed Dorado et al. (2011) have shown that improved sampling should increase nestedness. Clearly this is a contentious issue, and one which we do not address here.

There appears to be a combination of evidence justifying the validity of each potential driving force of nestedness, and it seems unlikely that, even if one is dominant, the others would have no effect (Vázquez et al., 2005, 2009b). Indeed, Olesen et al. (2008) saw a link between nestedness and abundance as well as nestedness and phenophase length (which is a result of complementarity/forbidden links). Stang et al. (2007) found a strong link between specialization asymmetry and nectar-holder size (a phenotypic trait) followed by abundance. Although this does not necessarily result in a similar link to nestedness, as nestedness implies specialization asymmetry but not *vice versa* (Bascompte and Jordano, 2007; Jordano et al., 2006), it is a promising sign. Our approach here is compatible with the concept of abundance, and indeed demonstrates a mechanism by which the effects of relative species abundances might lead to nestedness (see Chapter 3), but we cannot here consider the effects of sampling. Suffice to say that we attend to the pattern of nestedness, relying on the above mentioned studies as a proof that patterns of nestedness are indeed significant in host-parasite networks, and are unlikely to occur due to sampling error or abundance alone.

We therefore propose a model host-parasite network driven by trait coevolutionary trade-offs that includes a combination of influences from abundance, forbidden links and trait-matching, as it seems likely that evolutionary history and ecological factors together shape the structure of networks (Bascompte and Jordano, 2007; Blüthgen et al., 2007). Each species in our host-parasite system is assumed to have limited resources to use in interacting with all species from the opposite group (parasites or hosts respectively). These resources are focused on the transmission of infection, with parasites aiming to increase transmission while hosts attempt to decrease transmission. In Chapter 3, an exploratory model was proposed for a four-species system, with two hosts and two parasites. Here, this is expanded to a system with m hosts and

n parasites, while remaining identical in its core principles. Results are then given for $m = n = 5$. The aim of this is to discover whether the simpler model seen in Chapter 3 can explain patterns of nestedness in a larger system.

This chapter begins by outlining the model of Chapter 3 and describing some of the difficulties encountered in adapting it to a higher dimension. This is followed by a discussion of a series of evolutionary simulations and their resulting nestedness. Using the temperature and NODF metrics we found nestedness to occur more often than expected when trade-off shapes were weak (convex), while "anti-nestedness" occurred more often than expected when the majority of trade-off shapes were strong (concave).

4.2 The Model

This model uses a susceptible-infected system, with m host species and n microparasite species. S_i refers to susceptible hosts of species i, while I_{ij} refers to hosts of species i infected by parasite species j, where in this instance $i \in \mathbb{N}_m$, $j \in \mathbb{N}_n$. We then have

$$\begin{aligned} \frac{dS_i}{dt} =& \alpha_i M_i - \sum_{j \in \mathbb{N}_n} a_{ij} c_{ij} S_i F_j^i - \omega_i S_i M_i, \\ \frac{dI_{ij}}{dt} =& a_{ij} c_{ij} S_i F_j^i - \gamma_{ij} I_{ij} - \omega_i I_{ij} M_i. \end{aligned} \tag{4.2.1}$$

The model birth (α_i) and death (ω_i) rates depend on the host species i, and there is an additional infection-related death term; death rate γ_{ij} of host species i due to parasite species j. $M_i = S_i + \sum_{j \in \mathbb{N}_n} I_{ij}$ represents the total population size of host species i.

The force of infection F_j^i of parasite species j on host species i is given by

$$F_j^i = \sum_{k \in \mathbb{N}_m} \beta_j^{ik} I_{kj},$$

where β_j^{ik} is the pairwise potential infectious contact rate for the transfer of parasite j from host k to host i. In our model, this is moderated by the strategies adopted by the parasite j and the host i involved. It is then effectively given by $G_j^i = a_{ij} c_{ij} F_j^i$, with $0 \leq a_{ij} \leq 1$, $0 \leq c_{ij} \leq 1$. Here a_{ij} is a parasite phenotypic trait defining the relative probability of success of parasite j's attack on host i, and c_{ij} is a host phenotypic trait defining the relative probability of failure of host i's defence against parasite j. Parasites

benefit from high values of a_{ij}, while hosts benefit from low values of c_{ij}, reflecting the relative success or failure of transmission.

Species do not, however, have unlimited resources with which to ensure transmission or defence. Hence, our trade-off assumes that each parasite species j has a fixed amount of resource to allocate to infection of different host species i, where $i \in \mathbb{N}_m$. The trade-off is a species-specific function of interaction trait values, which is not dependent upon the population or environment. Similarly, a host species i varies strategy c_{ij} in order to reduce transmission of parasite j, where a similar trade-off is presumed. The transmission of infection to a susceptible host is now dependent on the force of infection and the relevant strategies which both host and parasite have adopted. As the trade-off shape can have an important effect on evolution (Best et al., 2009; Kisdi, 2006; Rueffler et al., 2006), we have assumed a trade-off shape here which can be altered to be either strong or weak, determined by a species-specific power (θ_j for parasite species j and ϕ_i for host species i). We have that

$$\sum_{i\in\mathbb{N}_m} a_{ij}^{\theta_j} = 1 \quad \text{and} \quad \sum_{j\in\mathbb{N}_n} c_{ij}^{\phi_i} = 1. \tag{4.2.2}$$

Note that $a_{ij} \in [0,1]$ and $c_{ij} \in [0,1]$ for all i, j. For parasite species j, $\theta_j < 1$ implies a strong trade-off, and $\theta_j > 1$ implies a weak trade-off. A parasite is a perfect generalist if $a_{ij} = (\frac{1}{m})^{\frac{1}{\theta_j}}$ for all i, which is henceforth termed the *neutral point*. At this point all trait-values for that parasite are equal, so it is investing equally in targeting all host species. Note, however, that different levels of defence by the host species may affect the relative success of the parasite in each species. The parasite is a complete specialist in host k if $a_{kj} = 1$ and $a_{ij} = 0$ for all $i \neq k$. In this case, it invests all of its resources in one host species only. Host species trade off their resources in a similar manner. Because species cannot coinfect a host, any infected host is unavailable to other parasites of all species, reducing the susceptible class. Hence, parasite species compete to infect any shared host species. A detailed analysis of the model where $m = n = 2$ can be found in Chapter 3.

4.3 Analysis

We used the technique of adaptive dynamics to analyse the model, which assumes that rare mutations occur, producing mutants with phenotypic traits

marginally different from those of residents. These mutants can only invade the population if their growth rate is positive in an equilibrium environment of residents. In that case, the population of mutants has the potential to grow in size over ecological time until the mutant phenotype becomes dominant and excludes residents (Dieckmann and Law, 1996; Geritz et al., 1998; Metz et al., 1996). Events are thus separated into evolutionary and ecological time-scales (Drossel and McKane, 2005). Chapter 3 gives a full analysis for a smaller system. Here, though, mutations occur between two trait-values during any evolutionary time-step; as a species' trait value with respect to an opponent increases due to mutation, the trait value with respect to one other opponent chosen at random must be reduced, as a consequence of the constraint in Eq. 4.2.2. Investigations using a full trade-off between all traits, as used by Dieckmann and Law (1996), were unable to satisfy this constraint. Therefore, evolution was partitioned into steps, and in each step a mutant of one random species with a slight variation in two random traits was introduced to the system (satisfying the invasion conditions given in Chapter 3). A successful mutant then replaced the resident, and the next evolutionary step was calculated at the new equilibrium containing the mutant. We used Gillespie's direct algorithm (Gillespie, 1977), which relies on stochastic events and the individuals within a population. This algorithm simulates the master equation stochastically, using a Monte Carlo procedure. A population of individuals is taken, and a list of all possible events (mutations of different species) is made along with the probabilities of such events occurring. The rate at which any event occurs is then calculated, and hence the time until the next event is simulated. Which specific event occurred is randomly chosen according to the probabilities, and both the time and populations are updated accordingly. The process is then repeated (see Keeling and Rohani, 2008). An example of the process of evolutionary change in trait values with time where $m = n = 3$ is given in figure 4-2. In this example, the final interaction matrix (of relative population densities) is given by U_1, and can be rearranged to give U_2, where

$$U_1 = \begin{pmatrix} 8 & 1 & 0 \\ 8 & 0 & 0 \\ 0 & 8 & 8 \end{pmatrix} \text{ and } U_2 = \begin{pmatrix} 0 & 8 & 8 \\ 8 & 1 & 0 \\ 8 & 0 & 0 \end{pmatrix}, \tag{4.3.3}$$

where columns represent parasites and rows hosts. Comparing this to figure 4-1(c), we see that this displays specialization asymmetry, but not nestedness.

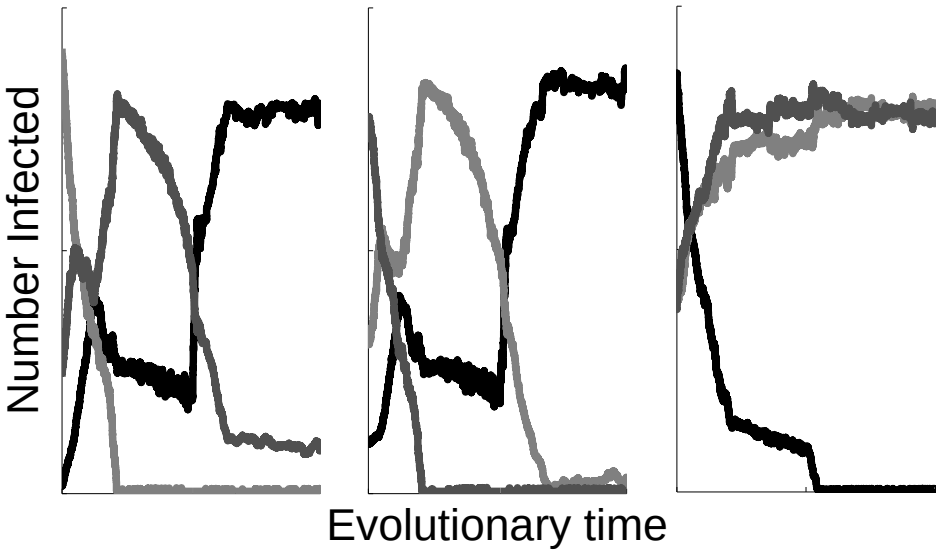


Figure 4-2: Example of the evolution of trait values in a system with 3 hosts and 3 parasites. Each line follows the evolution of the combined trait values of a specific host and parasite, to give the relative level of infection. Plots represent different hosts, and line shades different parasites. For example, the first plot represents the infection levels of all 3 parasites in the first host. The darkest (black) lines follow the infection levels of one particular parasite across all 3 hosts. The final infection levels, after a burn-off period, are used to create a matrix of species interactions, from which the nestedness is calculated.

To simulate this system, a range of trade-off types were investigated, and in each case a sample of matrices resulting from different initial trait-values were calculated. The initial trait-values of the system were important in calculating evolutionary change, producing a range of different results. Unfortunately, the space of all possible initial trait-values was very large. We therefore defined the initial host trait-values to display equal investment in each parasite species, and chose parasite initial trait-values using rejection sampling. A population size matrix was constructed using the resulting evolutionary trait-values after a set period of time, when a pattern emerges as the system reaches a noisy, but stable, end-point. This burn-off period was established using test simulations. Note that, potentially due to the lack of an imposed structure, which ensures that parasites or hosts could mutate trait values away from a potential threat, no species extinctions were ever witnessed. Whereas in Chapter 3 trait values were investigated for nestedness, here the size of a population infected with a particular parasite was used, with the number of infected hosts representing the population size. The resulting matrix was then tested for nestedness using the WNODF program of Almeida-Neto and Ulrich (2011) to measure nestedness according to the temperature index of Atmar and Patterson (1995, 1993), the discrepancy metric (Brualdi and Sanderson, 1999), the NODF metric (Almeida-Neto et al., 2008) and the weighted WNODF metric (Almeida-Neto and Ulrich, 2011).

Tests for nestedness yielded Z-scores obtained according to the following metrics: discrepancy, temperature, NODF and WNODF. Results included the Z-score and whether or not that Z-score was significant for each network at the 5% level. In order to discover whether our results demonstrated nestedness, we tested them as follows. Firstly, due to the random null models we compared the data to, in order to detect nestedness, we would expect 5% of matrices to be both unusually nested and unusually anti-nested (or cold and hot respectively, depending on the metric) (Joppa et al., 2010). In order to discover if more networks than expected fell into the range of unusually nested or anti-nested, we used a Chi-square test to compare the expected values. If we discovered that a significant number of networks were unusual, then we used a binomial test to discover whether there was a significant difference between the number of matrices that were unusually nested and those that were unusually anti-nested. This was repeated for a range of trade-off shapes, including weak trade-offs only, strong trade-offs only, a mixture with

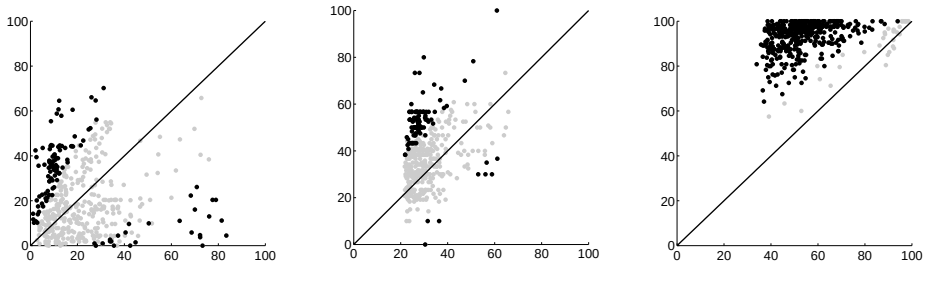
two weak and three strong trade-offs and *vice versa*.

4.4 Results

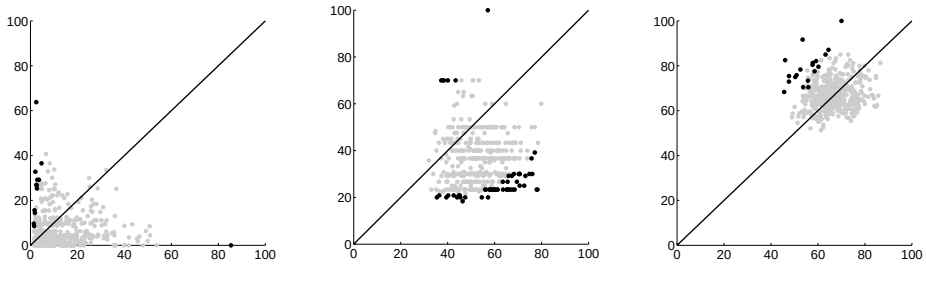
Our results are all given here at the 5% level of significance. In figure 4-3 we see for a range of trade-off strengths and metrics whether the resulting matrices were significantly different from null models. Here, black dots represent matrices that were significantly different, while grey dots represent those that were not. The line indicates the diagonal, where the observed and expected values for matrices were equal. Any matrices to the right of the diagonal, therefore, were more nested than expected, while any to the left were less nested.

We see in table 4.1 that in almost every case with every metric there was an unusually high number of non-random networks. The temperature, NODF and WNODF metrics were generally in agreement, with a low temperature occurring at the same time as significantly nested structures, and a high temperature occurring simultaneously to anti-nestedness. Interestingly, the discrepancy metric showed that not a single combination of trade-off strengths

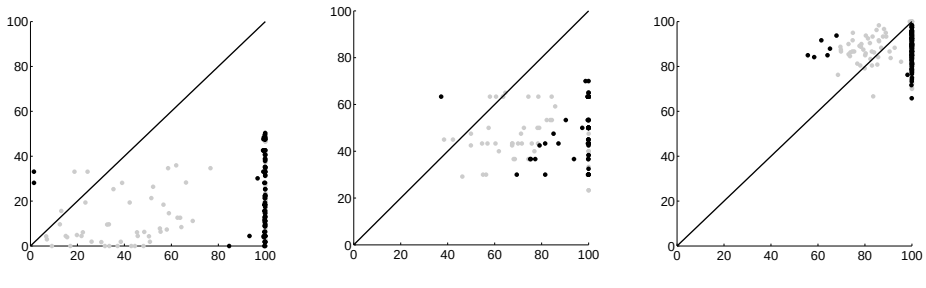
Figure 4-3 *(following page)*: A comparison of three of the metrics used to test for nestedness in systems with different strengths of trade-off in resources for parasite species. The x-axes here are the mean measurements (according to each specific metric) for 10 000 null models. The y-axes are the measurements taken from the observed matrices. Each column of sub-figures considers a different metric; plots (a, d, g, j) use the temperature metric (Atmar and Patterson, 1993), plots (b, e, h, k) use the NODF metric (Almeida-Neto et al., 2008) and plots (c, f, i, l) use the WNODF metric (Almeida-Neto and Ulrich, 2011). Each row of plots represents trade-offs of a different strength. Trade-off strengths are strong for all parasite species in plots (a–c), strong for a predominant proportion of parasite species in plots (d–f), weak for a predominant proportion of parasite species in plots (g–i) and weak for all parasite species in plots (j–l). We have re-scaled the metrics NODF and WNODF so that they may be compared to the temperature metric. Hence, for all three metrics, a value of 0 indicates perfect nestedness, while a value of 100 indicates "anti-nestedness." Black dots represent those matrices for which the Z-score ($\frac{Obs-Exp}{StDev_{Exp}}$) is significantly different from zero at a 5% level of confidence (Almeida-Neto and Ulrich, 2011). Grey dots represent those matrices for which the Z-score is not significantly different from zero. The solid line is the line $x = y$, and hence any points below the line are more nested than expected, while those above are less nested than expected.



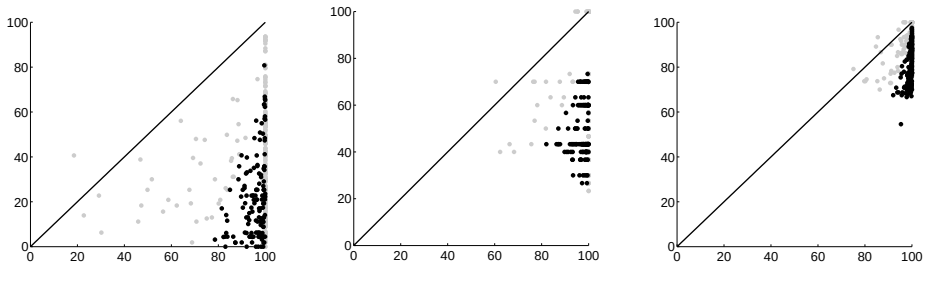
(a) (b) (c)



(d) (e) (f)



(g) (h) (i)



(j) (k) (l)

	Trade-off							
Metric	Strong		Mixed strong		Mixed weak		Weak	
	sig	sign	sig	sign	sig	sign	sig	sign
Disc	✓	−	✓	−	✓	−	✓	−
Temp	✓	−	✗	0	✓	+	✓	+
NODF	✓	−	✓	+	✓	+	✓	+
WNODF	✓	−	✗	0	✓	+	✓	+

Table 4.1: Table of results for different trade-off strengths under different metrics. Metrics include discrepancy (Brualdi and Sanderson, 1999), temperature (Atmar and Patterson, 1993), NODF (Almeida-Neto et al., 2008) and WNODF (Almeida-Neto and Ulrich, 2011). Trade-off strengths are either strong for all parasites, a mixture which is predominantly strong or predominantly weak, or weak for all parasites. One column (sig) indicates whether or not the data contained an unusually high number of non-random networks according to each metric (at a 95% level of confidence), and a second (sign) whether any unusual results were significantly positive or negative. Note the discrepancy and temperature metrics are rescaled such that, in agreement with the NODF and WNODF metrics, a positive Z-score indicates nestedness.

resulted in nestedness. There was always an unusual high number of matrices which had significantly high discrepancies, indicating a lack of nestedness (Brualdi and Sanderson, 1999). This could be due to the null model used, which increases the chances of incorrectly failing to reject the null hypothesis of no nestedness.

Ignoring the discrepancy metric, we see that if all parasite species have a strong trade-off, then each metric finds an unusual number of significantly anti-nested or hot networks. If all parasite species have weak trade-offs, or in fact if three out of five of parasites have weak trade-offs, then all three metrics (ignoring the discrepancy metric) find an unusually high number of significantly nested networks. If only two out of five of parasites have a weak trade-off, and the other three strong, however, then the results are more complicated. Here, both temperature and WNODF found that there were unusually fewer significantly nested or anti-nested networks than expected by chance. The results were random, with both less nestedness and anti-nestedness than expected by chance. The NODF metric, on the other hand, still found an unusual number of significantly nested networks.

4.4.1 Branching

We used, as motivation for our model, the work of Cobey et al. (2010), which looked at different strains of influenza virus binding via sialic acid receptors to a host's cell-surface oligosaccharides. Different hosts have different proportions of oligosaccharide types; waterfowl, horses and dogs contain Neu5Acα(2,3)-Gal linkages, humans and cats contain Neu5Acα(2,6)-Gal linkages and pigs and chickens possess both linkage types (Cobey et al., 2010). Cobey et al. (2010) modelled viruses which could adapt to bind preferentially to one linkage type or the other.

In our model we have expanded on this idea, presuming that each separate host species in our system has a specific linkage type, and a parasite must adapt to these. In addition, each host contains antibodies specific to each separate parasite species, which reduces the chances of an infection being successful. These antibodies are independent of the receptor type that the host contains, which does not evolve. We make no further assumptions with regards to species types or relationships, and have assumed equal transmission between all host species (dependent upon the respective host's antibody levels and the parasite's propensity for that host), although intraspecies transmission is presumed to be higher than interspecies transmission.

When we consider the manner in which species branching occurs, therefore, we must bear the above in mind. We assume that, if a host species branches in its trait values, this refers to the manner in which it produces antibodies for each parasite, which is independent of its receptor type. Therefore, although the host population now takes on two strategies with distinct trait values, parasites still target each strategy in an identical manner.

From the parasite perspective, the hosts are seen as an average of both strategies in proportion to their abundance. Similarly, if a parasite species branches in respect to which cell-surface oligosaccharides it binds to, although this results in a species with two separate strategies, we assume that the host species reacts to the average when its antibodies evolve. Because we look at mutations occurring in separate time steps (see Cobey et al., 2010 and Law et al., 2001) we can look at the monomorphic case only, where one species is evolving with respect to two traits only. This species then either goes extinct, replaces the resident or coexists with it (Kisdi, 2006). However, we only consider the branching of parasite species here, as sexual populations require geographical isolation or assortative mating in order for branching to

occur (Dieckmann and Doebeli, 1999).

Full speciation, therefore, cannot occur in our model, as species trait-branching only results in different strategies. In addition to this, species extinction is never been witnessed, due to the lack of structure. This ensures that a parasite species may always adapt to target one host or another, and it is not feasible for every single host to target one specific parasite, as this leaves them open to exploitation from other, more abundant parasites. The addition of a structure to the system, by limiting transmission pathways, could alter this, by leaving parasites in "dead-end" host species. Figure 4-4 shows an example of a system with 3 host species and 3 parasite species in which branching occurs. We see that the number of branching events increases with host mutation rate.

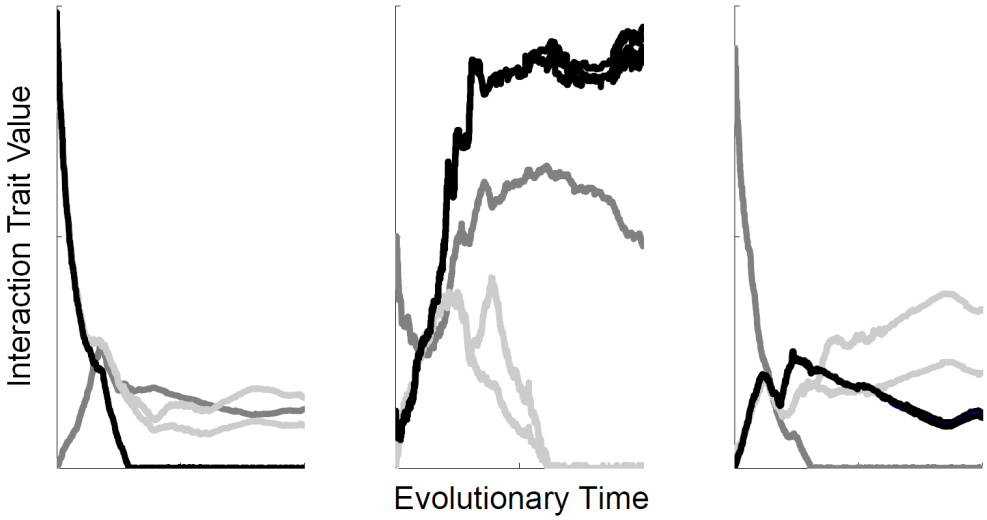


Figure 4-4: A plot of the evolution of trait values with time in a system with multiple hosts and parasites. Each plot represents a different host, and each colour of line a different parasite species. The x-axis is evolutionary time, and the y-axis the combination of each parasite's trait value for that host and the host's trait value for that parasite. In the first plot, therefore, if black represents interactions with parasite one, then the black lines represents the values of $a_{11}c_{11}$ for different strains of the same parasite species.

4.4.2 Structure

It is probable that extinctions do not occur due to the lack of structure in the system, as mentioned above. In order to test this, we impose different structures on a 3x3 system. The structures are outlined in figure 4-5 below,

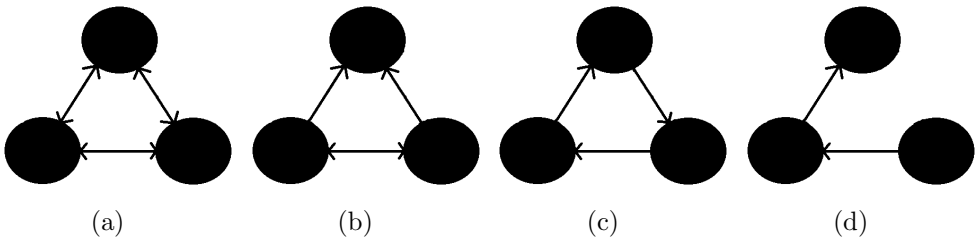


Figure 4-5: Outline of different transmission structures for a 3x3 host-parasite network. Transmission occurs between host species (circles) along transmission routes (arrows), and becomes increasingly more constrained from (a)-(d). Note, also, that both figure (b) and (d) have transmission in one direction only, and contain no cycles.

and the resulting stochastic evolutionary dynamics in figure 4-6. We expect that the directionality of predator-prey interactions will affect transmissability (Rossiter and Sukhdeo, 2011), and make assumptions accordingly. We see that, if transmission occurs only in one direction between hosts, then interaction traits are greatly constrained, and we see either parasite specificity (figure 4-6(b)), or indeed extinction (figure 4-6(d)). This serves to emphasize the importance of food web structure on host-parasite dynamics, as it is only through the imposition of a structure on transmission routes that we see species extinctions. In Chapter 7 we therefore investigate the importance on parasite species richness of host network dynamics within a food web.

4.5 Discussion

These results seem to be indicative of a fairly strong trend, where most forms of trade-off yield very strong structural patterns. If trade-offs are weaker, then the system is highly likely to be nested, whereas the more strong trade-offs there are the more likely the system is to exhibit anti-nestedness. This is in reasonable agreement with Joppa et al. (2010) and Poulin and Guégan (2000), who found, in host-parasite and -parasitoid webs, that there were an unusually high number of non-random networks, which contained both significantly nested and significantly anti-nested webs. In addition to this, Joppa et al. (2010) even suggested that the anti-nestedness might be due to coevolution, as our data suggest. It seems, then, that our proposed model could indeed drive patterns of nestedness and anti-nestedness in host-parasite food webs, a concept which is corroborated by a number of different metrics.

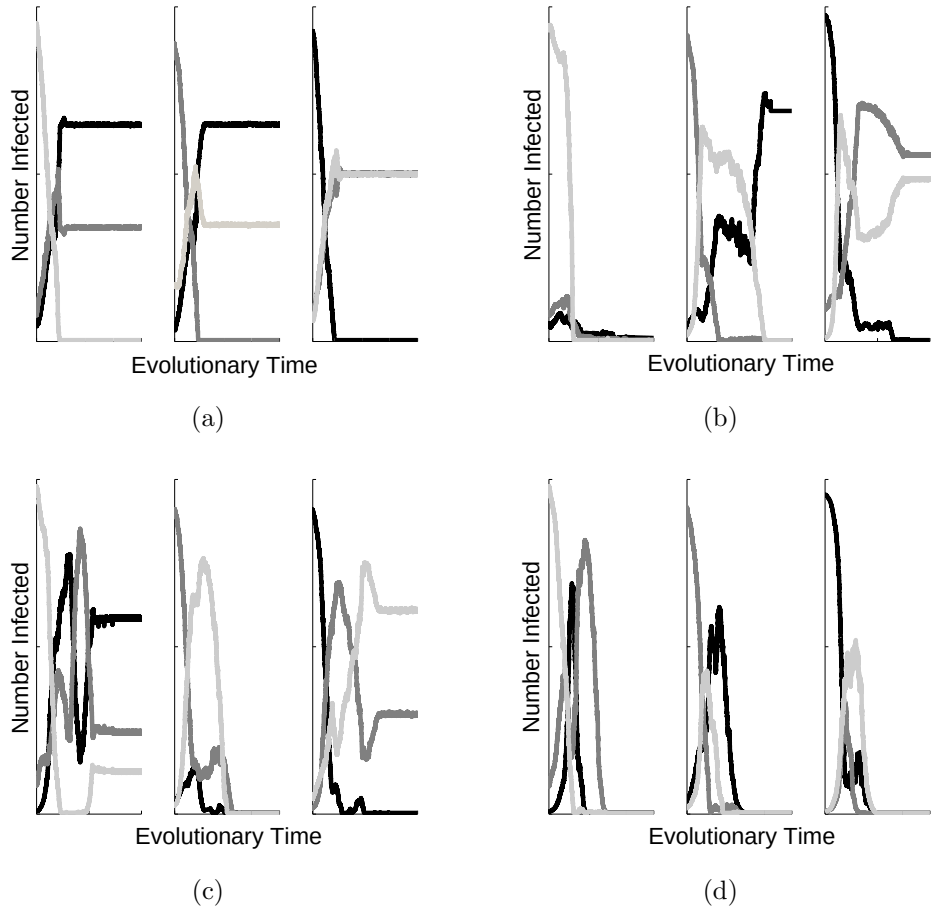


Figure 4-6: Evolutionary dynamics for host-parasite interactions with increasing constraints on transmission from (a)-(d). Each set of subfigures corresponds to the respective structure given by the subfigures in figure 4-5. In each subfigure there are three plots, each representing one host species. Each line follows the evolution of the combined trait values of the host and a particular parasite interacting with that host species to give relative levels of infection; each line colour represents a different parasite species.

The agreement of these results over a range of metrics is promising, supporting our conclusions. The failure of the discrepancy metric to agree with all except one of our results suggests that it may be an inappropriate metric to use, possibly due to the size of the networks, where nestedness is much less likely to be evident in both real-world and model networks (Guimarães Jr and Guimarães, 2006; Joppa et al., 2010; Santamaría and Rodríguez-Gironés, 2007). In small networks, the influence on nestedness of different species with an identical number of interactions is felt, promoting the possible incorrect acceptance of a null hypothesis of no nestedness (Almeida-Neto and Ulrich, 2011; Araujo et al., 2010a; Podani and Schmera, 2012). The remaining metrics appear less affected by this, and seem fairly interchangeable. The most reliable metric is likely to be the weighted WNODF metric, which considers interaction strengths, through population size, rather than binary data. It is, however, comforting that this metric agrees with both the temperature and NODF metrics, which have been used in the majority of empirical studies.

One important aspect to consider is the meaning of the term anti-nestedness. Here we simply use this to describe the results obtained according to the metrics used, but the actual physical representation of anti-nestedness may take on different forms depending on the metric and its authors' definitions. Almeida-Neto et al. (2007) describe the manner in which, for example, the term anti-nestedness has been used to describe random networks (Wright et al., 1998), checkerboard patterns (Duponte et al., 2003) and species absence from richer sites (Poulin and Guégan, 2000), amongst others. Here, to save confusion, we simply refer to anti-nestedness according to the metric.

Unlike Chapter 3, where specialization asymmetry was more evident in a system with strong or mixed trade-offs, our results show that in a larger system it is the presence of weak trade-offs that promotes nestedness. A caveat to this, however, is that even here we still analysed only a very small network. This should lead to lower than expected levels of nestedness (Guimarães Jr and Guimarães, 2006), and could significantly affect results. Unfortunately, due to computational times and the variety of possible initial trait-values (which have a significant effect on results, see Chapter 3), investigating larger networks was not feasible. In addition to this, altering the mutation rates of hosts compared to parasites (as in Chapter 3) might also suggest useful avenues for experimental research, predicting the circumstances under which we would expect to see nestedness. Again, however, computational constraints

severely limit this.

We propose here that the manner in which species make the best use of their limited resources leads to patterns of nestedness in host-parasite webs. This is influenced by both abundance and phenotypic trait-matching. Indeed, as this involves the evolution of trait values, matching may in fact arise as a consequence of these trade-offs (see, for example, Blüthgen et al., 2007). Vital to this is the aspect of coevolution, without which there could be no dynamical matching of traits. Although host evolution has often been ignored in the past, due to longer generation times and smaller populations, it is becoming clear that even low rates of evolution for hosts can have an effect on evolutionary outcomes (Best et al., 2009). This chapter demonstrates that the coevolution of multiple species might, in fact, be key to understanding the structure and function of inter-species relationships. Unfortunately, it also highlights the importance that chance can have in such large systems. For example, in figure 4-3(a) it appears that a system can evolve to be either significantly nested or significantly anti-nested depending on the initial trait-values (in relation to evolutionary repellers) and the stochastic evolutionary process, demonstrating that suitable repetition and care is required when investigating the coevolution of large networks.

The quantification and understanding of nestedness stretches beyond just one simple measure, as many network properties and patterns are inter-related (Burgos et al., 2007; Fortuna et al., 2010). Hence, the use of a dynamical model explaining one such pattern leaves many open questions concerning others, and any predictions that the model makes regarding them. There is a scarcity in research of dynamical models containing many species, and these could in fact be key to explaining nested trophic links (Montoya et al., 2006). This nested structure, of a core of species responsible for the majority of interactions, and an asymmetry in interactions, may be vital for the robustness of food webs (Jordano et al., 2006). Having demonstrated the application of trade-offs to host-parasite webs, it would be beneficial to repeat this for mutualistic webs, which have much higher levels of nestedness and no anti-nestedness (Joppa et al., 2010). For a host-parasite system, however, our trade-off concept can be used to explain nestedness, predicting a significant number of nested networks if most parasite trait trade-offs are weak, while predicting a higher number than expected of anti-nested networks if most trade-offs are strong.

CHAPTER 5

Higher Dimension Mutualistic System

Both ecological and evolutionary timescales are of importance when considering an ecological system; population dynamics affect the evolution of species traits, and *vice versa*. In Chapters 3 and 4, these two timescales have been used to explain a structural pattern in host-parasite networks, where the evolution of the manner in which species balance the use of their resources in interactions with each other was examined.

Patterns of both nestedness and anti-nestedness have been observed significantly more often than expected due to chance in host-parasite networks. In contrast, mutualistic networks tend to display a significant degree of nestedness, but are rarely anti-nested. Within networks with different interaction types, therefore, there is a feature promoting non-random structural patterns, such as nestedness and anti-nestedness, depending on the interaction types involved.

Here, we invoke the coevolution of species trait-values when allocating resources to interactions to explain the structural pattern of nestedness in a mutualistic community. We look at a bipartite, multi-species system, in which the strength of an interaction between two species is determined by the resources that each species invests in that relationship. We then analyse the evolution of these interactions using adaptive dynamics.

We found that the evolution of these interactions, reflecting the trade-off of resources, could be used to accurately predict that nestedness occurs

significantly more often than expect due to chance alone in a mutualistic network. This complements previous results applying the same concept to an antagonistic network. We conclude that population dynamics and resource trade-offs could be important promoters of structural patterns in ecological networks of all types. These affect aspects such as the network's long-term stability and reaction to change, as well as fluctuations in population sizes and interactions.

Nestedness, in which the interactions of specialist species form subsets of those of more generalist species, appears to be an important structural feature of many ecological networks (Sugihara and Ye, 2009). In a bipartite ecological community such as a plant-pollinator network, this means that the set of insect species pollinating a specialist plant will form a subset of those pollinating more generalist plants (Bascompte and Jordano, 2007).

This pattern is evident in many ecological networks (Carney and Dick, 2000; Flores et al., 2011; Graham et al., 2009; Kondoh et al., 2010; Lewinsohn et al., 2006; Thébault and Fontaine, 2008). We focus, however, on mutualistic networks, where there is strong evidence for the occurrence of nestedness (Bascompte et al., 2003; Bezerra et al., 2009; Guimarães Jr et al., 2006; Joppa et al., 2010; Jordano et al., 2006 although see Dicks et al., 2002; Poulin, 2007a). In mutualistic networks there is evidence for both specialization asymmetry, where specialists interact with generalists (Stang et al., 2007; Vázquez and Aizen, 2004), and connections between highly connected species (Melián and Bascompte, 2002). The combination of these two patterns also implies the presence of nestedness (Krishna et al., 2008).

Many theories have been proposed to explain patterns of nestedness seen across networks of different interaction types, including such forces as species abundance and the matching or mismatching of phenotypic traits (Bascompte and Jordano, 2007; Krishna et al., 2008; Lewinsohn et al., 2006; Olesen et al., 2008; Stang et al., 2007). A more abundant species is hypothesized to interact with a greater number of other species, leading to nestedness (Vázquez et al., 2009a). Similarly, nestedness could occur due to the preference for species entering a community for interactions which impose a lower competitive load, most likely to be interactions with generalists (Bastolla et al., 2009; Olesen et al., 2008). In comparison, phenotypic matching can be seen in examples such as differences in habitat range between plants and pollinators (Jordano et al., 2006), or the matching of a flower's nectar holder size and shape with

the length of a pollinator's tongue (Jordano et al., 2003). Both the matching of these two traits, or their complete mismatching to create forbidden links (where species are unable to interact at all), can determine the number of interactions that a species has with other species, again influencing the nestedness of a system (Jordano et al., 2003; Law et al., 2001). In addition to this, coevolution and evolutionary history are also important ecological factors, and have been postulated to lead to nestedness (Krishna et al., 2008).

Given the evidence for both these and other theories, it is often difficult to identify one factor alone that leads to nestedness. A possible alternative is that a combination of factors is important, with past evolutionary history, abundance and phenotypic traits all driving nestedness (Bascompte and Jordano, 2007; Lewinsohn et al., 2006; Olesen et al., 2008; Santamaría and Rodríguez-Gironés, 2007; Stang et al., 2007, although see Vázquez and Aizen, 2003). In this way, a network's structure and dynamics are linked (Bastolla et al., 2009).

In host-parasite networks, both more significantly nested and anti-nested networks than expected are seen (Joppa et al., 2010). In Chapters 3 and 4 we suggested that these patterns of nestedness and anti-nestedness could be driven by the coevolution of a trade-off in resources with which a species interacts with other species (see also Flores et al., 2011; Kondoh et al., 2010). In this way, species adaption alters the interaction network structure over time (see Kondoh, 2003 for the application of this to foraging efficiency). The concept of resource trade-off incorporated the ideas of both abundance and trait-complementarity in its architecture, and hence looked at both neutral and directed evolution, predicting that this could indeed drive structural patterns in host-parasite networks.

Here, we extend the idea to mutualistic networks, to investigate whether it is indeed successful at predicting patterns of nestedness regardless of the interaction type involved. Individuals have limited resources for interaction, particularly in consumer-resource interactions (into which category mutualisms fall) where the consumer has to use time and energy in order to find and consume the resource. In our model, mutants which use these resources more carefully or wisely will have higher growth rates and a greater chance of success. Over evolutionary time this will affect their interactions, altering the network structure and leading to networks with particular structural properties. The model is analysed using adaptive dynamics to follow the coevolution

of this resource use on interactions between species, and the resulting networks are tested for nestedness. We find that networks which begin with a random set of interactions do evolve to become significantly nested more often than expected due to chance alone, supporting the theory of Chapter 4 that a trade-off in interaction strengths drives patterns of nestedness in interaction networks; both host-parasite and mutualistic.

5.1 The Model

Unlike well-defined models for infectious diseases (Kermack and McKendrick, 1927), mutualistic interactions have been the focus of many different types of model, and there appears to be little agreement on a single general model structure (see Bastolla et al., 2009; Brauer and Castillo-Chávez, 2001; Brauer and Soudack, 1985; Dean, 1983; Holland et al., 2002; Law et al., 2001; Okuyama and Holland, 2008; Thébault and Fontaine, 2010). Specifically at higher dimensions, where there is more than one mutualistic interaction, models vary in their portrayal of events. Here we describe the model that we have focused on, and how it is expanded to a multi-dimensional system. Parameter values were based on Thébault and Fontaine (2010) for an obligate system and Bastolla et al. (2009) and Okuyama and Holland (2008) for a facultative system, with alterations made to account for the presence of species "traits" discussed below, and such that the asymptotic states are feasible, comparable equilibria in all cases.

Our model has two types of mutualistic species; plants (P_i) and animals (A_j). Each species fills a different niche in the system, and so does not interact with other species of the same type; plants may only interact in a mutualistic manner with animals, and *vice versa*. The model has the potential for both specialist and generalist plants and animals, interacting with different numbers of species of the opposite group. The population dynamics for a system with N plant species and M animal species are given by

$$\frac{dP_i}{dt} = \nu_i P_i - \zeta_i P_i^2 + \sum_{j=1}^{M} \frac{\epsilon_{ij} t_{ij} \tau_{ij} P_i A_j}{1 + \sum_{i=1}^{N} t_{ij} \tau_{ij} P_i}, \tag{5.1.1}$$

$$\frac{dA_j}{dt} = \upsilon_j A_j - z_j A_j^2 + \sum_{i=1}^{N} \frac{e_{ij} t_{ij} \tau_{ij} P_i A_j}{1 + \sum_{i=1}^{N} t_{ij} \tau_{ij} P_i}. \tag{5.1.2}$$

Natural growth rates are given by ν_i and v_j for populations of plant species i and animal species j respectively, whilst $\zeta_i P_i$ and $z_j A_j$ are density dependent death rates. The growth rates may be positive or negative, depending on whether the mutualism is a facultative or obligate one respectively (Brauer and Castillo-Chávez, 2001). This will have an effect on the levels of specialization in the system, with obligate mutualisms being associated with an increase of overall specialization (Blüthgen et al., 2007).

The final terms in equations 5.1.1 and 5.1.2 above describe a saturating response in the growth rate of plant species i due to the presence of animal species j and the response of animal species j to plant species i respectively (Holland et al., 2002). The saturation due to the plant species in the denominator of the growth rate from mutualism for both plants and animals reflects the fact that, in a foraging bout of one unit time for one particular animal, the probability of an encounter is determined by the relative densities of different plant species and not the animal. The term ϵ_{ij} represents the conversion efficiency for plant species i when gaining reward due to an interaction with animal species j. Similarly, e_{ij} represents the conversion efficiency for animal species j with respect to plant species i.

Thus far, our model is an unremarkable model of a network of mutualistic interactions. Now, however, we introduce the concept of a trade-off in resources. Similarly to the host-parasite models of Chapter 3 and Poitrineau et al. (2003), plants and animals allocate resources to increase their per-capita population growth rate due to the presence of one or the other of the opposite species. This is achieved by varying trait values τ_{ij} for plant species i with respect to animal species j (a measure of its investment in structures to attract and reward animals) and t_{ij} for animal species j with respect to plant species i (a measure of its time and energy investments when searching for plants, see Holland et al., 2002). These trait values then determine whether there is an interaction between the two species, and if so the strength of that interaction (Okuyama and Holland, 2008). An example of these traits in a plant could be nectar holder design; adjusting this increases the plants interactions with some pollinators and decreases its interaction strength with others (Vázquez and Aizen, 2004). However, we define the traits used to interact with different species as being unlinked, except in that they require the use of a limited set of resources. Therefore, each trait is specific to a species interaction, and its increase leads to an increase in interactions with that species only.

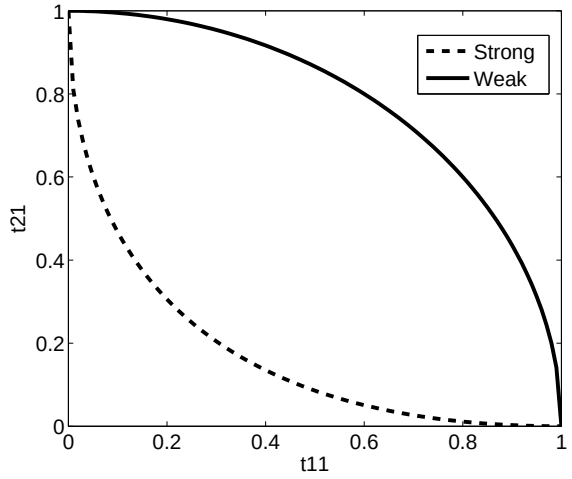


Figure 5-1: Examples of trade-off shapes for trait values of animal species 1 in a system with 2 plant species, where the trade-off is either strong ($s_1 = 0.5$) or weak ($s_1 = 2$)

There is a species-specific trade-off in the trait values for a species, independent of the population and environment (Kisdi, 2006). This is constructed here as follows:

$$\sum_{j=1}^{M} \tau_{ij}^{\sigma_i} = 1 \text{ for each } i, \text{ and } \sum_{i=1}^{N} t_{ij}^{s_j} = 1 \text{ for each } j, \tag{5.1.3}$$

where trade-off shapes are determined by σ_i, $s_j > 0$ for plant species i and animal species j respectively. Values for a trade-off shape greater than 1 represent a weak trade-off, while values less than one represent a strong trade-off (see figure 5-1).

For example, if $(N, M) = (2, 2)$ and $\sigma_1 = 1$, we have two plant and two animal species, with a linear trade-off for the traits of plant species 1. When $\tau_{11} = 0.5$ then $\tau_{12} = 0.5$, and plant species 1 invests equally in a mutualistic relationship with both animal species. If, on the other hand, $\tau_{11} = 1$, then $\tau_{12} = 0$ and plant species 1 invests solely in animal species 1, and does not interact at all with animal species 2.

An actual interaction term depends on the investments by both species involved in that interaction. As an example, a pollinator may not invest a great deal of time in visiting a particular plant species (so the trait value for the animal with respect to the plant species is low) but when it does so, it may find that the plant species is particularly easily accessible to it (so the trait value for the plant with respect to the animal is high), both factors

which then affect the strength of the interaction.

5.2 Method

We follow the coevolution of trait values, and hence interaction strengths between species, until they converge to a noisy coevolutionary stable strategy, where the network structure of interactions is fixed (although the weights of interactions may still evolve). The nestedness of the final binary interaction matrices is calculated and compared to null models of the same dimension and fill, using two metrics for nestedness; the temperature metric (Atmar and Patterson, 1993) and the NODF (Nestedness based on Overlap and Decreasing Fill) metric (Almeida-Neto et al., 2008).

Our simulation uses adaptive dynamics (Dieckmann and Law, 1996; Geritz et al., 1998; Metz et al., 1996), which assumes a one-to-one mapping of genotype to phenotype (Law et al., 2001). We presume that a rare mutant with a slightly different phenotypic trait value to a population of residents at equilibrium (determined by equations 5.1.1 and 5.1.2) is introduced to the system (Dieckmann and Law, 1996; Kisdi, 2006). The growth rate of this mutant population in the system reflects the success of the mutant trait, which is able to replace the resident population of that particular species if it can successfully invade. Hence, if a mutant species can invade and out-compete a resident, then it is presumed to replace the resident population and reach a nontrivial equilibrium on an ecological timescale, assumed to be much shorter than the evolutionary scale on which mutations occur (Law et al., 2001). This replacement of residents with mutants is known to be true for small phenotypic changes not near a population bifurcation or evolutionary equilibrium point, which covers our noisy system (Law et al., 2001). Note that all species produce mutants at an equal rate proportional to population density.

We therefore separate our problem into ecological and evolutionary timescales. In discrete evolutionary time, we use the Gillespie algorithm (Gillespie, 1977) to calculate the time between evolutionary events occurring. In each evolutionary event, a rare mutation of a random species is introduced to the system at a population equilibrium. One trait value of this mutant is randomly chosen to have mutated, and a second random trait value chosen to mutate in response, to ensure that equation 5.1.3 holds. This reflects the trade-off in species trait values; the increase of one trait must come at the

expense of a decrease of another. The invasion potential of the mutant is calculated according to equation 5.2.5 or 5.2.6 below as appropriate, and if the mutant successfully invades then it is presumed to replace the resident. The system containing the successful mutant is then presumed to reach an ecological equilibrium again before a new mutation event occurs in evolutionary time. We use an adaptive step-size Runga-Kutta method (Matlab, ode45) to follow the population dynamics. Over a sufficient period of time, the system approaches an asymptotic steady state, which is a local attractor.

5.2.1 Plant invasion conditions

For a mutant (E_i', P_i') of plant species i, where $E_i = (\tau_{i1}, \tau_{i2}, ..., \tau_{iM})$ reflects the plant's interaction trait values, with the resident population at equilibrium ($P_i = P_i^*, A_j = A_j^*$), the population growth rate is given by

$$\nu_i - \zeta_i P_i^* + \sum_{j=1}^{M} \frac{\epsilon_{ij} t_{ij} \tau'_{ij} A_j^*}{1 + \sum_{i=1}^{N} t_{ij} \tau_{ij} P_i^*}, \tag{5.2.4}$$

which follows directly from the population dynamics. Bearing in mind the equilibrium conditions for a non-trivial equilibrium, the invasion condition from equation 5.2.4 can be reduced to

$$\sum_{j=1}^{M} t_{ij}(\tau'_{ij} - \tau_{ij}) \frac{\epsilon_{ij} A_j^*}{1 + \sum_{i=1}^{N} t_{ij} \tau_{ij} P_i^*} > 0 \tag{5.2.5}$$

for successful invasion of the mutant.

5.2.2 Animal invasion conditions

The invasion conditions for a mutant animal are altered by the fact that the calculation of time spent searching is dependent on the mutant and its trait. This is unlike the case for plants, where an animal would spend the majority of its time encountering resident plants (and so the presence of mutant plants would not affect the denominator in equation 5.2.4). The growth rate of a mutant animal (D_j', A_j'), where $D_j = (d_{1j}, d_{2j}, ..., d_{Nj})$, reflects the animal's interaction trait-values, in a resident population ($P_i = P_i^*, A_j = A_j^*$) at equilibrium, is given by

$$v_j - z_j A_j^* + \sum_{i=1}^{N} \frac{e_{ij} t'_{ij} \tau_{ij} P_i^*}{1 + \sum_{i=1}^{N} t'_{ij} \tau_{ij} P_i^*}.$$

From this, and again considering the equilibrium conditions, the following condition for a mutant to successfully invade is obtained;

$$\sum_{i=1}^{N} \frac{e_{ij} t'_{ij} \tau_{ij} P_i^*}{1 + \sum_{i=1}^{N} t'_{ij} \tau_{ij} P_i^*} - \sum_{i=1}^{N} \frac{e_{ij} t_{ij} \tau_{ij} P_i^*}{1 + \sum_{i=1}^{N} t_{ij} \tau_{ij} P_i^*} > 0. \tag{5.2.6}$$

5.2.3 Calculating nestedness

The values of interest after evolution has been simulated are the final, noisy equilibrium trait values. These are used as a proxy for interaction strength, and hence allow us to construct a matrix of interactions. See figure 5-2 for an example. Note that species at levels of less than 0.01 are assumed to be extinct (a rare occurrence in our results) and interactions with a strength of less than 0.01 are similarly assumed to be zero. In the binary version of such matrices, columns represent animal species and rows plant species. An entry of 1 represents an interaction between the animal in that column and the plant in that row, while a 0 represents no interaction between the two species (Lewinsohn et al., 2006). In weighted networks, a matrix element is proportional to the strength of interaction between the two species. Here we use the weighted metrics that we obtain as final interaction matrices to define binary interaction matrices, as the systems in which nestedness has been observed in nature have often been binary (see, for example, Joppa et al., 2010). The final interaction strength given by figure 5-2, at a noisy coevolutionary stable strategy (discovered using simulations) is used to construct the binary interaction matrix below. The end points of the black lines, representing the evolutionary stable strategy of one animal species, are at zero and a value greater than zero. The binary forms of these are given by zero and one, representing no interaction with one plant species and an interaction with the second plant species. In comparison, the second animal interacts with both plant species (the grey lines are both greater than zero) giving us the complete interaction matrix

$$M_1 = \begin{pmatrix} 1 & 1 \\ 1 & 0 \end{pmatrix}, \tag{5.2.7}$$

where rows represent plant species and columns animals.

We simulate a system containing five plant and five animal species. This could be viewed as a module within a network, or a subweb within a whole food web, in which we would still expect to find nestedness (Bezerra et al.,

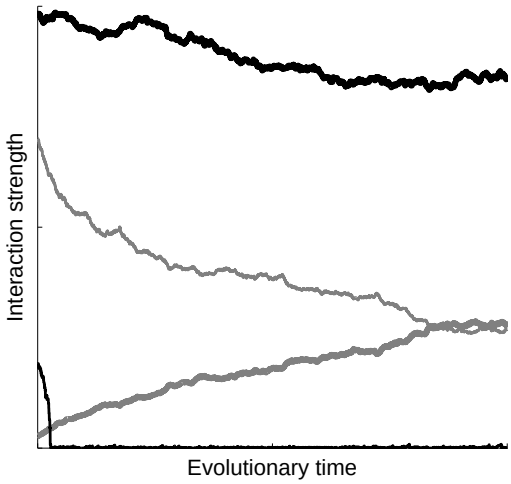


Figure 5-2: Examples of the change in interaction strengths due to the evolution of trait-values for a system with two plants and two animals, where the trade-off for both plants is linear, weak for one animal ($s_1 = 2$) and strong for a second ($s_2 = 0.5$). Each line demonstrates the evolution of the interaction strength for one particular plant-animal interaction. Different line shades indicate the animal species involved, and different line thickness the plant species. For example, black thick and thin lines indicate one animal species and its interactions with the two separate plant species.

2009). In general, there is an imbalance in the ratio of plant to animal species (Vázquez et al., 2009a), but our system is small and so we ignore this. Nevertheless, it is difficult to cover all of parameter space even for this system, given the large number of parameters, and so it is possible that other solutions exist to this model (Law et al., 2001).

Similarly to Chapter 4, the initial trait values for species affect the outcome (so the initial structure of interactions is influential in determining the final structure), and as there is a large state-space of initial traits for a 5x5 system, we use rejection sampling to randomly select the initial trait values. These are then allowed to evolve, as described above, following which they are tested for nestedness using two different metrics. These metrics calculate whether a matrix is more or less nested than expected due to chance alone when compared to 2000 null models with the same size, fill and row and column sums (Almeida-Neto and Ulrich, 2011).

5.3 Results

Figures 5-3-5-6 demonstrate our results for the nestedness of both facultative and obligate mutualisms measured using the temperature (Atmar and Patterson, 1993) and NODF (Almeida-Neto et al., 2008) metrics across a variety of combinations of trade-off strengths. Each set of plots looks at either a facultative or obligate mutualism, varying either plant or animal trade-off strengths. Each column of two plots investigates the nestedness of a system with a particular set of interaction strengths under two different metrics. For systems with strong/weak trade-off strengths, all animals (or plants, for figures 5-4,5-6) involved experienced a strong ($s_j = 0.5$)/weak ($s_j = 2$) trade-off on their interaction trait values. For systems with mixed trade-offs, different animal (or plant) species experience different trade-off strengths; some species in the system experienced a weak trade-off strength, and some a strong. In the first mixed case, the majority of animal (or plant) species experienced a strong trade-off, in the second a weak. In all cases, all plant (or animal) species experience a linear trade-off.

Note that the majority of significantly non-random networks occur below the diagonal, and hence are significantly nested. As the strengths in trade-off shapes decrease this is more and more evident. Using a Chi-square test to compare the expected numbers of significantly nested and anti-nested networks, all scenarios showed results which were significantly different from random, and in fact contained more significantly nested networks than expected due to chance alone. The only exceptions to this are using the temperature metric on obligate mutualisms where strong trade-offs for plants are present. We note that smaller networks, both empirical and theoretical, are less likely to display nestedness than larger networks (Guimarães Jr and Guimarães, 2006; Joppa et al., 2010; Santamaría and Rodríguez-Gironés, 2007). As we find much evidence for nestedness in our small networks here, this emphasizes the pervasive pattern of nestedness that our model suggests at in larger networks, which we were unable to simulate due to computational constraints.

Our only ambiguous result for facultative systems is for a linear trade-off for animals and a weak trade-off for all plant species. In this case, interaction matrices evolved to include all possible interactions, under which circumstances it is not possible to calculate nestedness. Hence, all points in figures 5-6(d) and 5-6(h) are at (0,0) or (100,100) respectively, depending on the

System		Trade-off							
		Strong		Mix strong		Mix weak		Weak	
		T	N	T	N	T	N	T	N
Fac.	Animal	+	+	+	+	+	+	+	+
	Plant	+	+	+	+	+	+		
Obl.	Animal	0	+	0	+	0	+	0	+
	Plant	+/−	+	+	+	+	+	+	+

Table 5.1: Table of results for different mutualistic systems, with different trade-off strengths under different metrics. Mutualistic systems are either facultative or obligate. Metrics include the temperature (T, see Atmar and Patterson, 1993) and NODF (N, see Almeida-Neto et al., 2008) metrics. Trade-off strengths vary for animal or plant species only in each row, and are either strong for all animals/plants, a mixture which is predominantly strong or predominantly weak, or weak for all animals/plants. A "+" symbol denotes the fact that there were more significantly nested networks than expected due to chance alone, a "+/−" that there were both more significantly nested and significantly anti-nested networks than expected, and a "0" that the distribution of nestedness across the networks appeared random according to that metric. Blank entries correspond to a scenario in which relative nestedness could not be calculated. Calculations used a Chi-square test.

metric. At these points, no matrices are significantly different to random and it is not possible to comment on their nestedness.

In the case of obligate mutualisms, the temperature metric does not find more nested networks than expected due to chance alone when varying animal trade-offs, but neither does it find more anti-nested networks than expected. Instead, the networks appear to be primarily random, according to the temperature metric. Indeed, the only case in which we see more anti-nested networks than expected due to chance alone is for an obligate mutualism with strong plant trade-offs. For a full compilation of results, see table 5.1.

Finally, in figure 5-7 we compare the observed nestedness for our results and the observed nestedness of matrices of the same fill where the presence of interactions is based on relative species abundances. We see that the NODF metric demonstrates that our results often yield higher than expected levels of nestedness compared to systems in which abundance determines interactions. On the other hand, the temperature metric demonstrates the opposite. This is possibly due to the obligate systems in our results, in which the temperature metric did not find a significant number of non-random matrices.

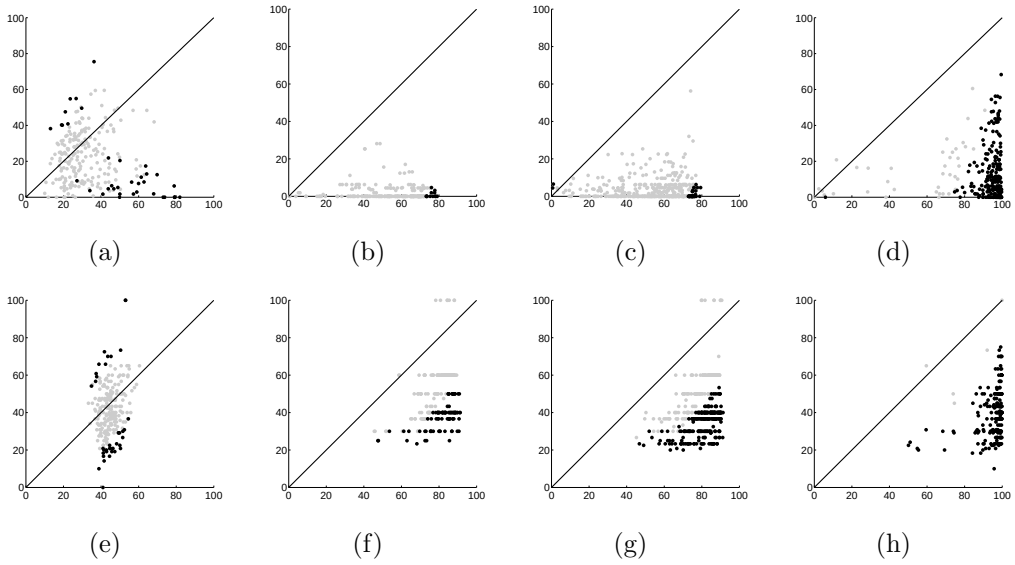


Figure 5-3: Two metrics used to test systems with different animal species trade-off strengths (plant species trade-offs are all linear) for nestedness after evolution in a facultative system, where the x-axes represent the mean measurements (according to each specific metric) for 2,000 null models and the y-axes the measurements for the observed final binary interaction matrices. The rows of subfigures use different metrics; plots (a-d) use the temperature metric (Atmar and Patterson, 1993) and plots (e-h) use the NODF metric (Almeida-Neto et al., 2008). The columns of plots contain networks with animal species trade-offs of different strengths. Trade-off strengths are strong for all animal species in plots (a, e), strong for a predominant proportion of animal species in plots (b, f), weak for a predominant proportion of animal species in plots (c, g) and weak for all animal species in plots (d, h). We have re-scaled the metric NODF so that it may be compared to the temperature metric. Hence, for both metrics, a value of 0 indicates perfect nestedness, while a value of 100 indicates anti-nestedness according to the metric. Black dots represent those matrices for which the Z-score ($\frac{Obs-Exp}{StDev_{Exp}}$) is significantly different from zero at a 5% level of confidence (Almeida-Neto and Ulrich, 2011). Grey dots represent those matrices for which the Z-score is not significantly different from zero. The solid line is the line $x = y$, and hence any points below the line are more nested than expected, while those above are less nested than expected. Parameter values for equations 5.1.1 and 5.1.2 are $\nu_i = 0.1$, $\zeta_i = 0.005$, $\epsilon_i = 0.1$ and $\tau_i = 1$ for all i, and $v_j = 0.5$, $z_j = 0.004$, $e_j = 0.9$ and $t_j = 1$ for all j.

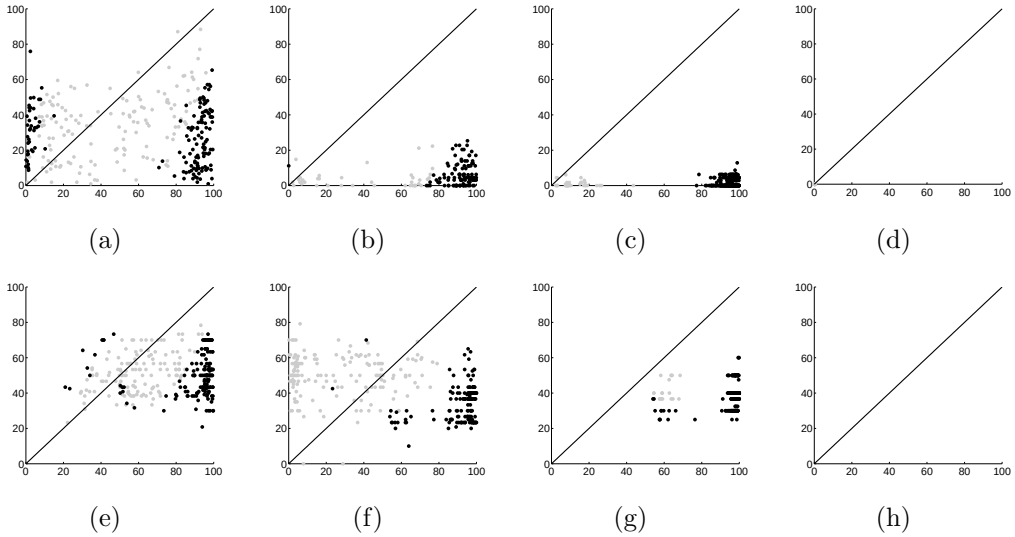


Figure 5-4: Systems with different plant species trade-off strengths (animal species trade-offs are all linear) for nestedness after evolution in a facultative system. The columns of plots contain networks with plant species trade-offs of different strengths. Trade-off strengths are strong for all plant species in plots (a, e), strong for a predominant proportion of plant species in plots (b, f), weak for a predominant proportion of plant species in plots (c, g) and weak for all plant species in plots (d, h). Parameter values are identical to figure 4-3

5.4 Discussion

This chapter uses a dynamical mathematical model which depends on species trait-values to recreate non-random patterns of nested mutualistic interaction networks, as opposed to networks of pairwise, compartmentalized interactions (Jordano et al., 2006). A nested architecture is important in many ways, as it can potentially promote lower connectivity, stability and biodiversity, as well as the persistence of specialist species (Bascompte and Jordano, 2007; Bascompte et al., 2003; Bastolla et al., 2009; Bezerra et al., 2009; Fortuna et al., 2010; Okuyama and Holland, 2008; Sugihara and Ye, 2009; Thébault and Fontaine, 2010; Zhang et al., 2011, although see James et al., 2012; Kondoh et al., 2010 for the causal relationship). Hence understanding the causal factors of nestedness could have far-reaching consequences. Here we have shown that coevolution, via the trade-off of resources contributing to interaction strength between species, can facilitate nestedness in a mutualistic network.

Across two different types of mutualism, using two different metrics, and a range of trade-off strengths for both animal and plant species, more networks than expected are significantly nested. This pattern does appear to be less

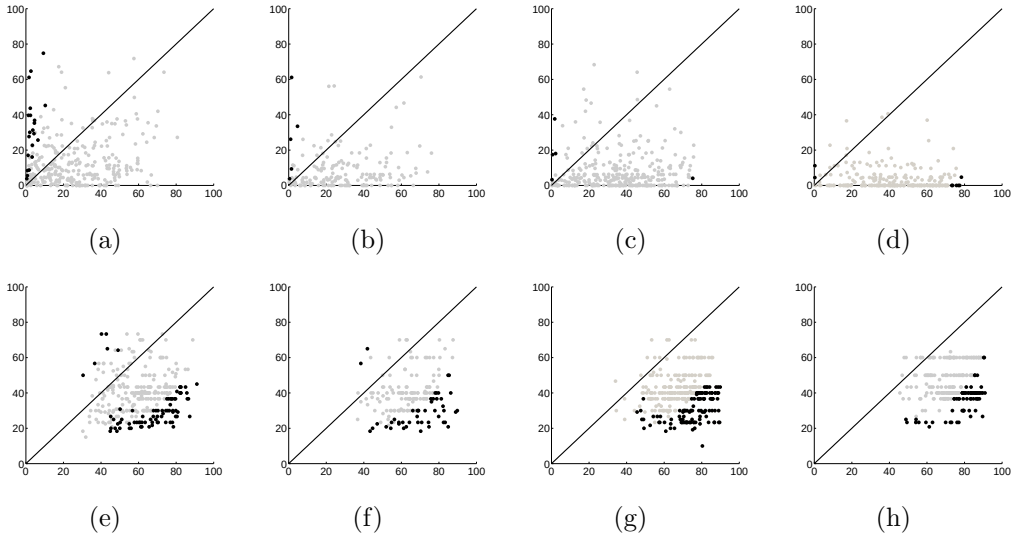


Figure 5-5: Systems with different animal species trade-off strengths (plant species trade-offs are all linear) for nestedness after evolution in an obligate system. The columns of plots contain networks with animal species trade-offs of different strengths. Trade-off strengths are strong for all animal species in plots (a, e), strong for a predominant proportion of animal species in plots (b, f), weak for a predominant proportion of animal species in plots (c, g) and weak for all animal species in plots (d, h). Parameter values are identical to figure 4-3, except for $\alpha_i = -0.1$ for all i and $a_j = -0.5$ for all j.

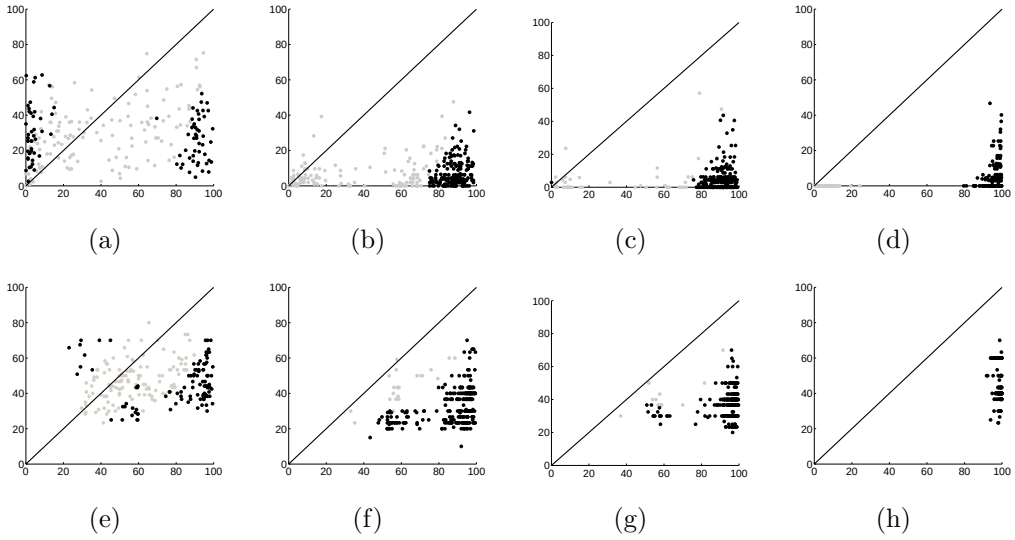


Figure 5-6: Two metrics used to test systems with different plant species trade-off strengths (animal species trade-offs are all linear) for nestedness after evolution in an obligate system. The columns of plots contain networks with plant species trade-offs of different strengths. Trade-off strengths are strong for all plant species in plots (a, e), strong for a predominant proportion of plant species in plots (b, f), weak for a predominant proportion of plant species in plots (c, g) and weak for all plant species in plots (d, h). Parameter values are identical to figure 5-5.

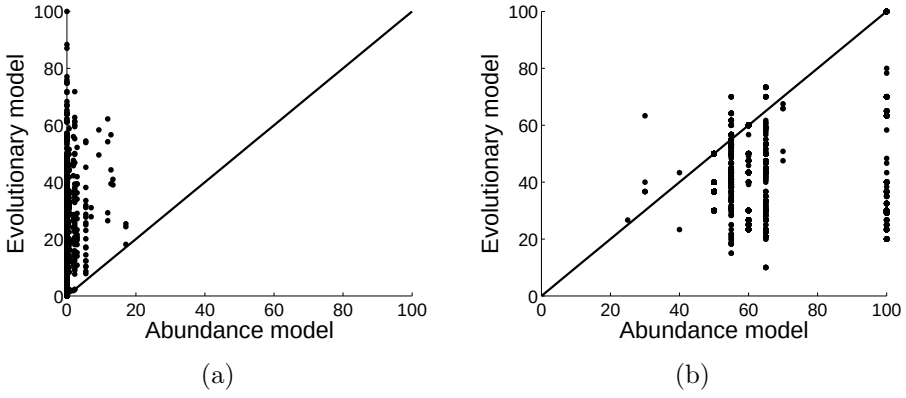


Figure 5-7: A comparison of the nestedness of matrices under our evolutionary model and when interactions are assigned to matrices of identical fill proportional to species abundance. Each figure represents a different metric; (a) the temperature metric and (b) the NODF metric. Note that the NODF metric has been rescaled such that, for both metrics, a measure of 0 represents complete nestedness, and a measure of 100 anti-nestedness according to that metric.

evident when all trade-off strengths are strong; although more networks than expected are significantly nested, there are some networks that are significantly anti-nested. This is unlike other scenarios where weak trade-offs are present, resulting in no anti-nestedness what-so-ever. However, there are still no more significantly anti-nested networks than expected due to chance alone, although the temperature metric finds that there are no more nested networks than expected. However, Ulrich et al. (2009) suggest that the NODF metric is more appropriate for interaction matrices than the temperature metric, and we see that this metric still finds a significant number of nested networks. The NODF metric finds more nested networks than expected due to chance across both mutualism types and all trade-off strengths for both species, and it never finds more anti-nested networks than expected.

Indeed, in only one case, for obligate mutualisms with a strong plant trade-off measured using the temperature metric, are there a significant number of anti-nested networks. This, in fact, serves to emphasize the ubiquity of significantly nested networks in our model, and that there are far fewer significantly anti-nested networks than expected due to chance when considering all of the networks in our results together. It also suggests possible lines of research to confirm our theory empirically; our model predicts that mutualistic networks that are anti-nested are likely to contain many strong trade-offs in species interaction traits, and are more likely to be discovered when using the temperature metric.

In comparison, in Chapter 4 we showed that a similar model resulted in a significant number of host-parasite networks being both more nested and anti-nested than expected due to chance alone. This model applied the concept of resource trade-offs in interaction strengths to a network where interactions were of a different type to here, and demonstrated that it did drive the formation of patterns observed in those systems. This concept, therefore, appears to match the empirical results of Joppa et al. (2010) very well (although see Kondoh et al., 2010); where nestedness occurs in mutualistic networks, and both nestedness and anti-nestedness in host-parasite networks. This demonstrates that network patterns could arise simply due to the evolutionary pressures on individuals within them. Factors such as species abundances, phenotypes and competitive load all combine to put pressure on resource use by a species, and it is through this combination of pressures that network structure evolves. Strikingly, our evolutionary model results in matrices that are more nested

than those based on abundance alone, when measured using the NODF metric, which confirms the importance of a number of interacting factors driving nestedness. Although the temperature metric contradicts this, we note again the suggestion of Ulrich et al. (2009) that this metric may be inappropriate for interaction matrices, and that the two metrics are in complete disagreement.

In a similar manner, Zhang et al. (2011) used partner interaction switching, in response to environmental conditions and the availability of resources, to account for nestedness in mutualistic networks. Our model does not require such extreme reactions from species, and instead looks at those which adapt slowly, as a result of random mutations and evolutionary rather than goal-driven change. Even small changes may be important to the nestedness of a system, with different individual species contributing in different manners (Joppa and Williams, 2011), and hence we focus on less extreme fluctuations. In addition to this, we do not require that the number of species interactions remain fixed, and instead approximate an interaction using the trait-values of both species involved. In this way, our model could be seen as more realistic, and reliant on evolutionary pressures.

An interesting extension of this model would be to alter the growth rates for species and the relative benefits that they offer. In our model, all species are assumed to be identical in these aspects, yet Zhang et al. (2011) proposed amendments in their model to determine whether it is simply the relative abundance of species that drives nestedness, or the benefits that each species provides to others. The contribution of individual species to the nestedness of a system also shows that those which contribute most strongly are also at most risk of extinction, and are detrimental to biodiversity (Saavedra et al., 2011). Therefore, although our model demonstrates that many potential causal factors of nestedness can be combined in a relatively straight-forward way, it could also be decomposed and used to assess the importance of the different factors.

5.5 Conclusion

Although our model leaves us with many question of the potential consequences that trade-offs in trait values could have, such as their effect on evolutionary stability, it does suggest that these natural trade-offs in how species use resources could be responsible for large-scale network patterns,

such as nestedness, across both host-parasite and mutualistic networks. The concept of resource trade-off demonstrates an important way in which the structure and stability of these systems can be seen to be intrinsically linked to their dynamics (see Melián and Bascompte, 2004). In this way, the roles of abundance and trait-values can simply be reduced to the optimum use of resources by different species. It seems that the evolution of this resource use can have far-reaching consequences, even predicting structural patterns in networks of species with very different interaction types.

In this work we have demonstrated the manner in which the population dynamics of a system can evolve to shape its structural properties, and hence alter both its stability and its complexity. When considering even an entire ecosystem, therefore, we suggest that its structure could depend on interactions at multiple time-scales, which can in part be traced back to simple evolutionary driving forces. Even large, complex structures of ecological interactions are dependent on the needs and pressures of their smallest members.

CHAPTER 6

Downward Asymmetry

We discuss now a brief outline of a second aspect of parasite presence, the structural feature of species richness in downwardly asymmetric interactions, and demonstrate how this could arise simply due to an increase in basic reproductive ratio for parasite species. This feature is particularly interesting, as it considers not just multiple host and parasite species, but in addition to this a network structure imposed on interactions between host species. Again, this expands our knowledge and experience of modelling parasites as a part of food webs.

A range of factors, including host body size, latitude and habitat, are all influential in determining non-random patterns of parasite biodiversity and abundance in different web types (Randhawa and Poulin, 2010). One of the most important factors from a network perspective, however, is the structure of interaction links in these webs (Chen et al., 2008; Marcogliese, 2002), which determines transmission routes for trophically transmitted parasites.

These are parasites that are dependent on transmission from a host prey species to a host predator species by consumption of the prey, in order to complete their life cycle. Once infecting the predator, the parasite relies on routes such as predator faecal contamination with parasite eggs to ensure that the life cycle continues with infection of a prey individual. Examples of such parasites can be found amongst cestodes, trematodes, nematodes and acanthocephalans, which often have a life-cycle that includes an intermediate

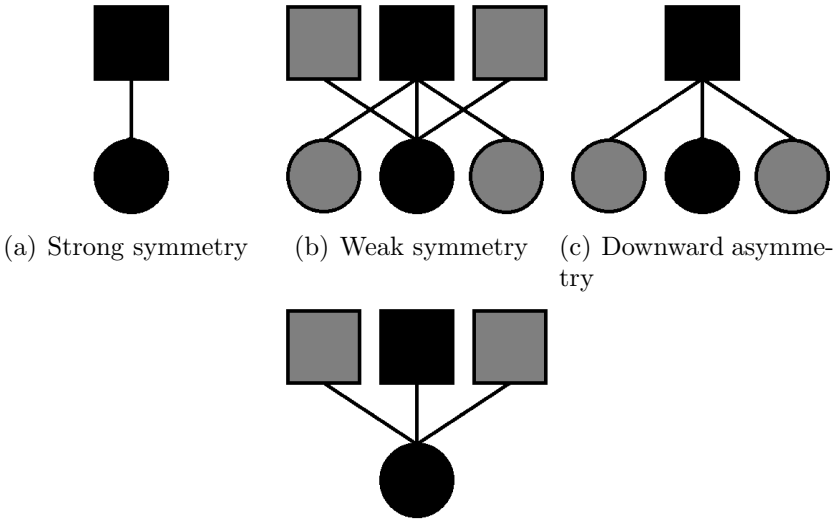
host that is prey to a definitive host (see, for example, Choisy et al., 2003; Dobson, 1988; Lafferty, 1999).

The transmission link via consumption of these parasites implies that the trophic level and position of a host are important for the diversity of its parasites (Paterson et al., 2012; Poulin and Leung, 2011), but a combination of other factors such as host diet range, vulnerability to predation and proximity to predators, prey and resources may also be influential (Anderson and Sukhdeo, 2011; Chen et al., 2008; Marcogliese, 2002; Paterson et al., 2012; Poulin and Leung, 2011). These all depend on the structure of interactions in a network that a food web describes.

Key to the factors described above, such as host diet range, are the number of predator and prey species involved in a subset of interactions; we outline the different interaction possibilities in figure 6-1, redrawn from Rossiter and Sukhdeo (2011). The number of competitors and predator or prey species that a host has alters potential transmission routes for parasites, potentially affecting the trophic parasite species density for hosts. Comparing the interaction motifs shown in this figure, we note that predator-prey interactions with many prey species, such as figure 6-1(c), are less likely to see population cycles, which increase extinction risk (Inouye, 1980), than strongly symmetric interactions with only a single prey species such as in figure 6-1(a). This stable environment is more conducive for trophic transmission of parasites (Anderson and Sukhdeo, 2011). In addition, interactions with multiple predator species may result in dilution (figures 6-1(b) and 6-1(d)); if only one predator species is a definitive host, then every time an infected individual is consumed by a different predator the parasite is lost (Rossiter and Sukhdeo, 2011, although see Chen et al., 2008).

Thus it can be argued that downwardly asymmetric interactions (one predator species with many prey species), which do not lead to dilution and are relatively stable, are theoretically best for the persistence of parasite populations. Rossiter and Sukhdeo (2011) recently demonstrated empirically that trophically transmitted parasites are funnelled towards downwardly asymmetric interactions; although the presence of more prey species could potentially provide more trophically transmitted parasites, downwardly asymmetric interactions contained even more parasites than expected due to this increase in prey.

In downwardly asymmetric interactions, intermediate hosts have few preda-



(a) Strong symmetry (b) Weak symmetry (c) Downward asymmetry

(d) Upward asymmetry

Figure 6-1: Types of predator-prey interactions in which trophic transmission of a parasite may occur. Black boxes represent predator (square) and prey (round) host species, and lines represent trophic interactions. Grey boxes represent non-host species. Redrawn from Rossiter and Sukhdeo (2011).

tors and definitive hosts have many prey species. The effects of dilution on parasite transmission have been discussed in detail before (see Ostfeld and Keesing, 2012; Rossiter and Sukhdeo, 2011), but not the effects of stable interactions with a greater number of prey species. Here we show that the basic reproductive ratio R_0, often considered a threshold for parasite persistence, is higher for parasites in such stable systems, demonstrating that the connection between interaction stability in downwardly asymmetric interactions and parasite presence is possibly due to the increase in basic reproductive ratio that results for such parasites.

In order to make this comparison between the values for R_0 in these different systems, we calculate the value for R_0 in both equilibrium and oscillatory multi-species environments, noting that the potential to do so accurately has only recently been realised. In oscillating systems, with multiple host species and periodic cycles such as those seen in the limit cycles of predator-prey interactions (Maynard Smith and Slatkin, 1971), calculating the basic reproductive ratio for a parasite is greatly complicated compared to equilibrium populations. One aspect of this regards the consideration in the R_0 calculation of an infected individual entering a wholly susceptible population. However, when population oscillations are present, the susceptible population

size varies depending on when the infected individual is introduced.

In recent years much work has been conducted in this area. Threshold calculations have been accomplished by using the unstable equilibrium population size of host species (Chattopadhyay and Arino, 1999), and for simple systems where only one species is infected, the time-averaged population size over one cycle, although this has been shown to be insufficient for systems with many host species (Bate and Hilker, 2013; see also Hsieh and Hsiao, 2008 for stability conditions). Another alternative for discovering the impact that parasites have in such systems is to focus on the forcing required to exclude parasites in periodic environments as a form of control (Greenman and Pasour, 2012).

Recently, however, Bacaër and others (Bacaër, 2009, 2007; Bacaër and Guernaoui, 2006; Rebelo et al., 2012; Wang and Zhao, 2008) have described a method with which to calculate R_0 in a periodic environment, interpreting it in a seasonal model as the "asymptotic ratio of total infections in two successive generations of the infection tree" in the linearization about the disease-free state (Bacaër and Ait Dads, 2012).

Taking this more complex approach, we use Floquet theory to calculate R_0 numerically. We then compare the values for R_0 in different interaction motif types, including strongly symmetric and oscillatory and equilibrated downwardly asymmetric interactions. We discover that parasites transmitted through downwardly asymmetric interactions in which a second prey species has acted to stabilise the system do indeed have a higher basic reproductive ratio than those transmitted through strongly symmetric interactions. However, if the addition of a second prey species does not stabilise the system, then the basic reproductive ratio of trophically transmitted parasites decreases.

6.1 The Model

We begin by describing a predator-prey system with infection, based on the work of Hadeler and Freedman (1989), although we include handling time of prey and infection-related mortality independent of increased vulnerability to predation. We propose a general theoretical model for a trophically transmitted parasite, where the parasite requires both an intermediate (prey) host and a definitive (predator) host, and there is no intraspecies infection. The

parasite is transmitted from prey to predator by consumption of infected prey species, and from predator to prey environmentally through routes such as faecal contamination. Infected individuals immediately become infectious, and remain so for life. Here we describe a model where Y represents the population of the prey species, and P the predator. The presence of a pathogen leads to infected classes y and p of prey and predator species respectively.

$$\begin{aligned}
\frac{dY}{dt} =&(Y+y)(b-dY) - \tau_y Y p - \frac{\gamma Y(P+p)}{H+h\gamma Y+vh\gamma y},\\
\frac{dP}{dt} =&\frac{\epsilon\gamma(P+p)Y}{H+h\gamma Y+vh\gamma y} - \delta P + \frac{\epsilon v\gamma(P+p-\tau_p P)y}{H+h\gamma Y+vh\gamma y},\\
\frac{dy}{dt} =&\tau_y Y p - d(Y+y)y - \omega_y y - \frac{v\gamma(P+p)y}{H+h\gamma Y+vh\gamma y},\\
\frac{dp}{dt} =&\frac{\tau_p \epsilon v\gamma y P}{H+h\gamma Y+vh\gamma y} - (\omega_p+\delta)p.
\end{aligned} \tag{6.1.1}$$

The net per capita production, in the absence of parasites, is given for the prey species by $b - dY$, with a handling time h for each prey with respect to the predator, and an interaction coefficient γ. H is a half saturation density for a Holling type II functional response. The predator converts prey to energy with efficiency ϵ, and dies in the absence of prey at a rate δ. The parasite is transmitted at rate τ_y from the predator to prey through the environment, and τ_p is the trophic transmission parameter for parasite transmission from the prey species to the predator upon efficient consumption. Infected prey are more vulnerable to predation depending on parameter v. Infected individuals experience infection-induced mortality at rates ω_y and ω_p for prey and predators respectively. Here we base parameter values on Bate and Hilker (2013) and Hadeler and Freedman (1989), such that there is a stable limit cycle in the absence of parasites: $b = 0.4$, $d = 0.01$, $h = 1.5$, $\gamma = 0.6$, $H = 3$, $\epsilon = 0.9$, $\delta = 0.5$, $\omega_y = \omega_p = 0$, $v = 1$, $\tau_p = 0.1$ and we vary τ_y.

In order to compare the values for R_0 when additional prey species are present, we also include non-host prey species for certain calculations. We follow the methods of Comins and Hassell and others (Comins and Hassell, 1976; McLellan et al., 2010; van Leeuwen et al., 2007) with species in different niches (although apparent competition may still occur, see Holt and Lawton, 1994). For species in identical niches, competition occurs, increasing the

density-dependent death term to include all prey individuals, as opposed to those of the same species only. Equations are identical to system 6.1.1 above, although the new prey species do not have an infection term, and the functional response includes the additional prey species. Details are given below for a total of k prey species:

$$\begin{aligned}
\frac{dY_1}{dt} =& (Y_1 + y)(b_1 - d_1 Y_1) - \tau_y Y_1 p - \frac{\gamma_1 Y_1 (P + p)}{H_1 + \sum_{j \in \mathbb{N}_k} h_j \gamma_j Y_j + v h_1 \gamma_1 y}, \\
\frac{dY_i}{dt} =& Y_i(b_i - d_i Y_i) - \frac{\gamma_i Y_i (P + p)}{H_i + \sum_{j \in \mathbb{N}_k} h_j \gamma_j Y_j + v h_1 \gamma_1 y}, \text{ for } i \in \{2, 3, ...k\}, \\
\frac{dP}{dt} =& \sum_{i \in \mathbb{N}_k} \frac{\epsilon_i \gamma_i (P + p) Y_i}{H_i + \sum_{j \in \mathbb{N}_k} h_j \gamma_j Y_j + v h_1 \gamma_1 y} - \delta P + \frac{\epsilon_1 v \gamma_1(P + p - \tau_p P) y}{H_1 + \sum_{j \in \mathbb{N}_k} h_j \gamma_j Y_j + v h_1 \gamma_1 y}, \\
\frac{dy}{dt} =& \tau_y Y_1 p - d_1 (Y_1 + y) y - \omega_y y - \frac{v \gamma_1 (P + p) y}{H_1 + \sum_{j \in \mathbb{N}_k} h_j \gamma_j Y_j + v h_1 \gamma_1 y}, \\
\frac{dp}{dt} =& \frac{\tau_p \epsilon_1 v \gamma_1 y P}{H_1 + \sum_{j \in \mathbb{N}_k} h_j \gamma_j Y_j + v h_1 \gamma_1 y} - (\omega_p + \delta) p. \qquad (6.1.2)
\end{aligned}$$

6.2 Analysis

In order to discover when a parasite is endemic, we calculate the basic reproductive ratio, R_0. Traditionally, R_0 has been thought of as the average number of newly infected individuals that one infected individual infects in a population of susceptibles (Anderson and May, 1979). An R_0 value at or above the threshold of unity implies that a parasite is able to persist in that environment (Rebelo et al., 2012; Wang and Zhao, 2008 but see Davis et al., 2008; Salkeld et al., 2010). Here we consider this to be the case, ignoring spatial and scale factors and focusing on the general theoretical framework, reviewed in Heesterbeek (2002).

We outline below the methods we use to find R_0 for a trophically transmitted parasite in two different systems; a multi-prey system at equilibrium (see, for example, Diekmann et al., 1990), and an oscillating predator-prey system.

Firstly, for the multi-prey equilibrium system, we note that the disease-free equilibrium must be obtained numerically. The functional response is dependent on the additional prey species (given by Y_j, where $j \in 2, 3, ...k$), and hence the next generation matrix (Diekmann et al., 2010) is given, for

species at steady states $Y_j = Y_j^*$, $P = P^*$, by

$$\boldsymbol{M} = -\boldsymbol{F}\boldsymbol{V}^{-1},$$

where

$$\boldsymbol{F} = \begin{pmatrix} 0 & \tau_y Y_1^* \\ \frac{\tau_p P^* \epsilon_1 v \gamma_1}{H_1 + \sum_{j \in \mathbb{N}_k} h_j \gamma_j Y_j^*} & 0 \end{pmatrix} \tag{6.2.3}$$

and

$$\boldsymbol{V} = \begin{pmatrix} -d_1 Y_1^* - \omega_y - \frac{P^* v \gamma_1}{H_1 + \sum_{j \in \mathbb{N}_k} h_j \gamma_j Y_j^*} & 0 \\ 0 & -\omega_p - \delta \end{pmatrix}. \tag{6.2.4}$$

From this we calculate the spectral radius of $\boldsymbol{M}$ to give R_0.

As mentioned above, it has been suggested that, for an oscillating system, using the time-average population over one cycle rather than the equilibrium population should yield R_0 in a system where only one species is a host (Bate and Hilker, 2013). However, Bacaër and Ouifki (2007) and Bacaër (2007) have demonstrated that using the time-averaged population may over- or under-estimate R_0 in a more complex scenario such as ours.

On the other hand, for a one-prey system with a disease-free limit cycle and time-dependent populations $P^*(t)$ and $Y^*(t)$, we may use Floquet theory to investigate the spectral radius of the "next-year matrix," measured over one period θ (see Bacaër, 2007). Where in the cycle the calculation begins is immaterial, yielding identical results for all initial values. R_0 is the unique, positive, real number such that the spectral radius of $\boldsymbol{Z}(t)$ at time $t = \theta$ is one, where

$$\frac{d\boldsymbol{Z}(t)}{dt} = \begin{pmatrix} -dY^*(t) - \omega_y - \frac{P^*(t) v \gamma}{H + h\gamma Y^*(t)} & \frac{\tau_y Y^*(t)}{R_0} \\ \frac{\tau_p P^*(t) \epsilon v \gamma}{R_0 (H + h\gamma Y^*(t))} & -\omega_p - \delta \end{pmatrix} \boldsymbol{Z}(t), \tag{6.2.5}$$

and

$$\boldsymbol{Z}(0) = \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix}. \tag{6.2.6}$$

Note that populations are time-dependent, and not constant. The period θ of the limit cycle is estimated using the Matlab program lomb.m, which calculates the most common period in a system using the Lomb normalized periodogram. We then compute R_0 numerically, using ode45 and a dichotomy method in Matlab. A guess is made for R_0, and the spectral radius calculated

accordingly. This is compared to the spectral radius for the previous guess, and a new informed guess made until the spectral radius is approximately one. The method is identical for an oscillating multi-prey system, except for the appropriate changes in indices and the functional response.

Note also that if $P^*(t)$ and $Y^*(t)$ are constant, then equation 6.2.5 can also be written as

$$\frac{d\boldsymbol{Z}(t)}{dt} = \left(\boldsymbol{V} + \frac{1}{R_0}\boldsymbol{F}\right)\boldsymbol{Z}, \tag{6.2.7}$$

from which we see the eigenvalue equation

$$\lambda\boldsymbol{u} = \left(\boldsymbol{V} + \frac{1}{R_0}\boldsymbol{F}\right)\boldsymbol{u}. \tag{6.2.8}$$

Now, setting $\lambda = 0$, for vector $\boldsymbol{v}$ such that $\boldsymbol{u} = \boldsymbol{V}^{-1}\boldsymbol{v}$,

$$0 = \boldsymbol{v} + \frac{1}{R_0}\boldsymbol{F}\boldsymbol{V}^{-1}\boldsymbol{v}. \tag{6.2.9}$$

We therefore see that R_0 is indeed an eigenvalue of $-\boldsymbol{F}\boldsymbol{V}^{-1}$, as given by the next-generation equilibrium matrix approach.

6.3 Results

In figure 6-2, we look at the value that R_0 takes on for systems with either one or two prey species occupying different niches over a range of transmission parameters. In a scenario with oscillating populations we use Floquet theory and in a scenario that reaches an equilibrium we use the next-generation equilibrium matrix approach. We note that these two methods are equivalent, as discussed above, and if we use equilibrium values numerically in the Floquet theory approach we obtain identical answers to the next generation equilibrium matrix (see Bacaër and Guernaoui, 2006 for more details). Analytically, we see that assuming the population is at disease-free equilibrium implies that the coefficient in equation 6.2.5 is not time-dependent. We require that the eigenvalue of this matrix corresponding to the greatest growth rate is zero, so that the spectral radius is also independent of time, from which we can solve for R_0. We find that this is identical to the R_0 taken from the next-generation equilibrium approach.

The scenario with only one prey species is taken to be oscillating, but we look at the addition of an extra prey with three different outcomes. In the

first case, the predator-prey system oscillates in the absence of either one of the prey species, and still oscillates when both are present (the additional prey species has $b = 0.3$, $h = 1.9$ and $H = 1.2$). We call this the oscillating two-prey system. The second case is similar, although there is competition, affecting the death rate, between prey species ($\delta = 0.4$, and the additional prey species has $d = 0.0105$, $h = 1.9$ and $H = 3.5$). This is the competitive two-prey system, and also displays limit cycles. In the third case, the predator-prey system oscillates in the absence of the second prey, while that second prey is unable to coexist with the predator in the absence of the first prey species (the additional prey species has $b = 0.5$, $h = 1.9$ and $H = 1.5$). However, when both prey are present, the system reaches a stable equilibrium. We call this the equilibrium two-prey system.

When calculating R_0 for the latter, in the absence of the first prey a parasite would not be able to exist, as there would be no predator to ensure trophic transmission continued as normal. However, when both prey are present as well as the predator, then the parasite can exist in either prey (and for our chosen parameter values it makes very little difference which). Note that a plot fixing $\tau_y = 0.1$ and varying τ_p is identical to figure 6-2, and the symmetry can be further observed in figure 6-5.

For comparative purposes, figures 6-3 and 6-4 demonstrate the population dynamics in each case for a prey transmission parameter of 0.6. In this case, a trophically transmitted parasite should only be able to exist in the case where there are two stabilising prey in a system at disease-free equilibrium. The parasite-free systems are initially allowed to reach an equilibrium, or periodic oscillations, after which a parasite is introduced. In the single-prey, oscillating and competitive two-prey systems, the parasite swiftly becomes extinct (after an initial peak of infections early in the cycle). In the equilibrium two-prey system, the parasite reduces to very low levels, before increasing to establish a long-term stable equilibrium.

Finally, we investigate the threshold for the different systems described above over varying values for both transmission parameters. Figure 6-5 shows our results.

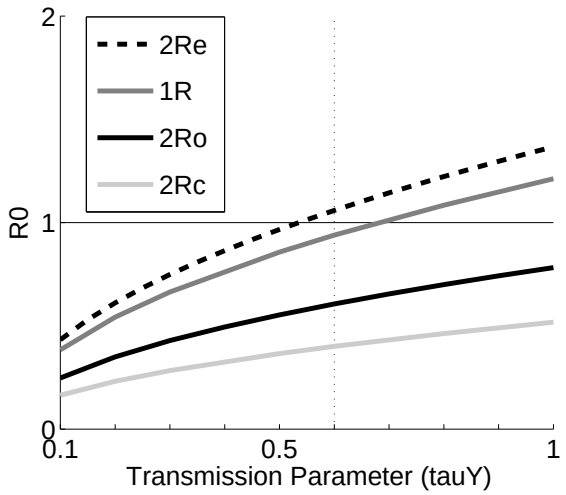
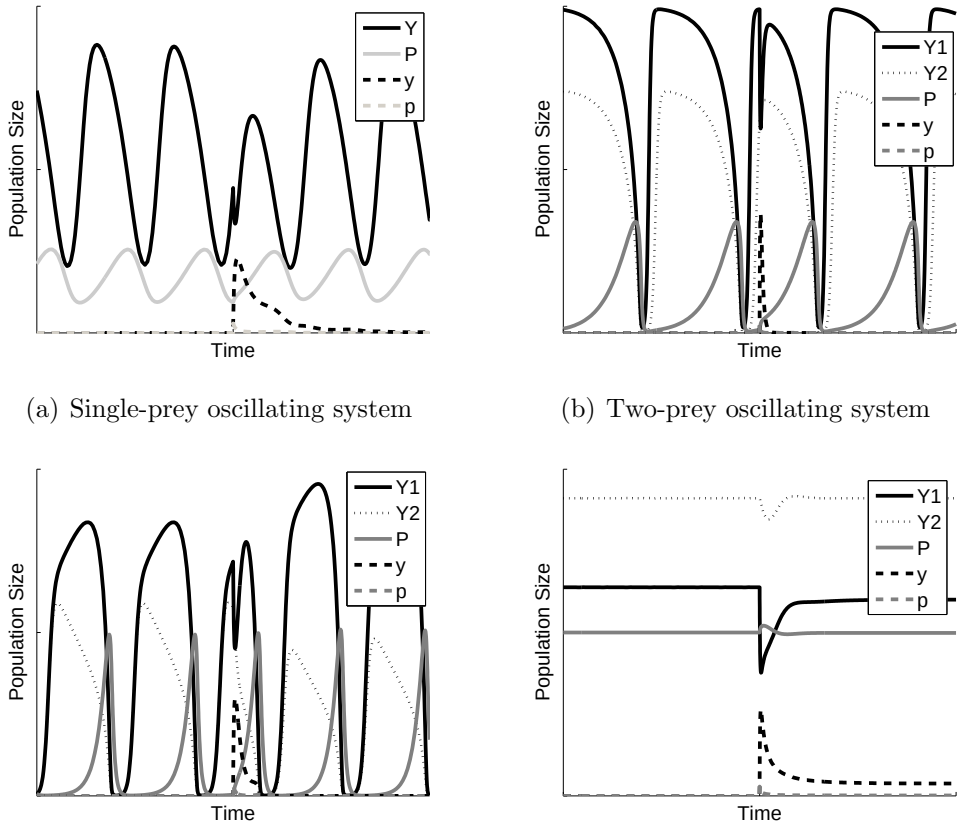


Figure 6-2: Comparing R_0 for systems with one prey only (1R), two prey in different niches where the system continues to oscillate in a limit cycle (2Ro), two prey in the same niche where the system continues to oscillate in a limit cycle (2Rc), and two prey where the additional prey has stabilised the system so that it has reached equilibrium (2Re), for varying τ_y. The vertical dotted line marks $\tau_y = 0.6$, where, in descending order of magnitude to four decimal places, our systems find $R_0 = 1.032$, $R_0 = 0.938$, $R_0 = 0.606$ and $R_0 = 0.401$ respectively.

6.4 Discussion

Previously, it was thought that the very concept of R_0 was not applicable to nonautonomous cases (Heesterbeek and Roberts, 1995), although whether that transferred to cases where periods were a result of population dynamics was not clear. Specifically, threshold values could be related to R_0 if all coefficients had a common period, but here all coefficients are constant (Zhang et al., 2008). In any case, both measures used above have been shown to be threshold quantities, and, in fact, give the basic reproductive ratio (Bacaër, 2007; Heesterbeek and Roberts, 1995; van den Driessche and Watmough, 2002). We can, therefore, compare the lowering of the threshold (and hence lowering of transmission parameters required for an epidemic) when a secondary prey species stabilises the system, and the increase of the threshold when a secondary prey species does not stabilise the system (and subsequent increase in required transmission parameters).

The appeal of downwardly asymmetric interactions to trophically transmitted parasites can be argued both verbally and empirically (Rossiter and Sukhdeo, 2011), but here we demonstrate this theoretically. The addition of



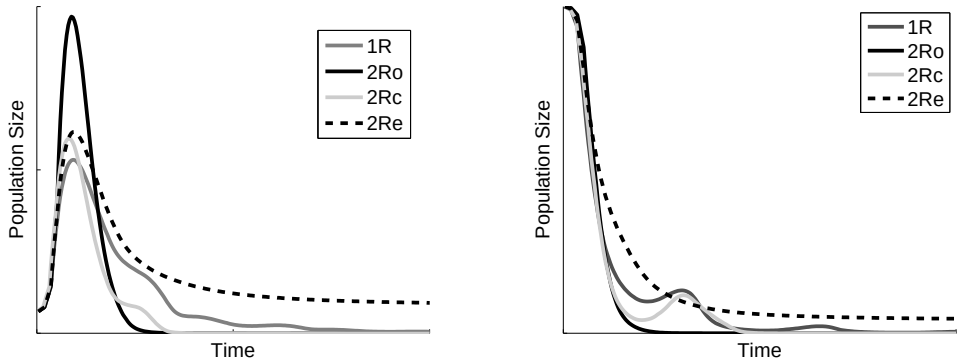
(a) Single-prey oscillating system

(b) Two-prey oscillating system

(c) Two-prey competitive system

(d) Two-prey stable system

Figure 6-3: Introducing a parasite to different predator-prey systems with identical transmission rates. Figure (a) shows a single-predator, single-prey oscillating system. Figure (b) shows an oscillating system with two prey species, and similarly figure (c) shows an oscillating system with prey in the same niche. In figure (d) we see a stable, equilibrated system with two prey species. For the cases with more than one prey species, we show the non-host prey dynamics as Y_2. After each system equilibrates, or settles into a periodic oscillation as appropriate, a trophically-transmitted parasite is introduced to one prey species. In figures (a), (b) and (c), after an initial peak in infections the parasite population decreases steadily to extinction, upon which the system returns to its oscillations. In figure 6-3(d) the parasite decreases to very low levels, but is able to persist in the population at an equilibrium. The infected populations are plotted in greater detail in figure 6-4, starting at the point of parasite introduction.



(a) Infected prey (b) Infected predator

Figure 6-4: Close-up of parasite dynamics after introduction to the systems described in figure 6-3. Figure (a) considers levels of infected prey, and figure (b) levels of infected predators in a system with one prey only (1R), two prey in different niches where the system continues to oscillate in a limit cycle (2Ro), two prey in the same niche where the system continues to oscillate in a limit cycle (2Rc), and two prey where the additional prey has stabilised the system so that it has reached equilibrium (2Re).

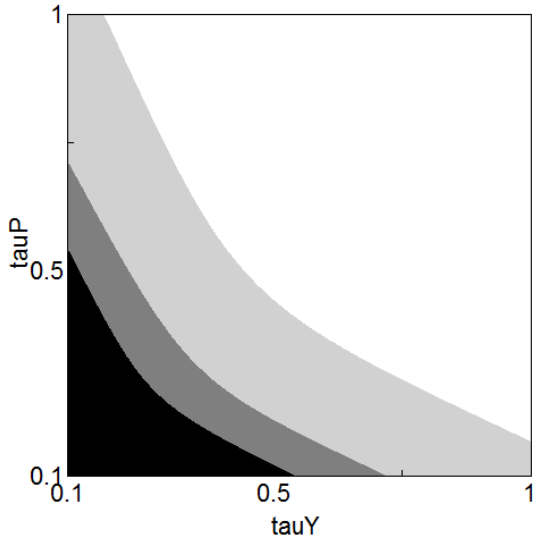


Figure 6-5: Threshold dynamics for varying transmission parameters in different systems. The x-axis measures transmission to prey, and the y-axis transmission to predators. In the black region, a parasite is unable to establish in any of the systems; the single prey, oscillating two-prey or equilibrium two-prey system. In the dark grey region a parasite can invade the equilibrium two-prey system, and in the light grey region it can invade both the single prey and equilibrium two-prey systems. In the white region all systems may be invaded by the parasite. We do not consider the competitive system here.

prey species to an oscillating predator-prey system, making it downwardly asymmetric, can be seen to decrease the transmission threshold for parasite persistence in scenarios where the additional species occupy different niches, and stabilise the oscillations in the system. Indeed, given that either prey species separately may now harbour the parasite, where when alone in the system the second prey was not able to coexist with the predator (and hence would be unable to harbour a trophically transmitted parasite), we are much more likely to see the persistence of trophically transmitted parasites in this system than in an oscillating system with either one or two hosts. This suggests an interesting avenue for research into parasite species richness in such systems, as a decrease in threshold for persistence could lead to an increase in species richness, although our exclusion of multi-parasite dynamics means that this work is out of the scope of our current model.

This is borne out by figure 6-5, where the stable system has the lowest threshold across all possible transmission parameters. In addition, the shape of the transmission threshold curve gives some insight into parasite dynamics. It can be seen that a parasite manipulating the behaviour of the intermediate host to increase τ_p is more likely to persist, as is often observed empirically through behavioural alterations (see, for example, Dobson, 1988; Lafferty and Morris, 1996). Additionally, increasing τ_y by increasing the lifetime of the parasite in the environmental stage could also lead to persistence. However, as the shape of this threshold curve is identical for each of our systems, we focus instead on the increase in parameter values that allow for parasite persistence.

We see that when a second prey is added to our system that does not stabilise it, then the value for R_0 decreases, reducing the likelihood that a parasite can persist. This occurs whether or not the prey species occupy the same niche, and in both cases results in the reduction of R_0. We would therefore expect even fewer parasites than in single-prey systems. This seems logical, as additional prey species competing with the host for the same niche could decrease host abundance and density, potentially lowering transmission of the parasite (which we assume can only infect the host prey species).

As additional prey species are added, it becomes increasingly difficult to identify parameters which lead to stable equilibria. This is, however, influenced by the functional response type, which could drastically alter results (Ferguson et al., 2012; Gibson et al., 2005; Holt and Lawton, 1994), and

indeed the larger structure of an entire web and its interactions could make these sub-systems more stable (Dambacher et al., 2002; Matsuda et al., 1996). Nevertheless, we are able to theoretically demonstrate that the addition of stabilising prey species to a system, unlike the addition of non-stabilising prey, does indeed increase the chances of parasite persistence in that system, which could have further consequences for parasite species richness.

CHAPTER 7

Network Stability

In Chapters 4 and 5 the importance of interaction type in a bipartite network can be seen. According to the theory discussed and developed there, a host-parasite network is more likely than expected due to chance to be non-random in structure, being either significantly nested or anti-nested, while a mutualistic network is more likely than expected to be significantly nested. In addition, in Chapter 6 the importance of interactions between host species on the persistence of a parasite can be seen. In this last chapter, we take these ideas further, considering the importance of the presence of parasites on networks of species interactions. We base our model on the work of Mougi and Kondoh (2012), and consider the effect of increased parasite species richness on the stability of model networks.

This is an area of much interest in the literature, where it is increasingly becoming clear that trophic food webs and networks with only one interaction type are unsatisfactory descriptions of species dynamics in an ecosystem (Fontaine et al., 2011; Kéfi et al., 2012). Real ecological networks are composed of species which interact ecologically and evolutionarily with one another in many different forms; as predators, prey, mutualists, competitors, hosts, parasites and so on. Considering one interaction type alone can fundamentally change the network architecture that favours stability (Thébault and Fontaine, 2010), and it is therefore necessary to consider multiple types simultaneously. Considering only a bipartite sub-network of a certain class

of interaction in isolation fails to capture the realities of species interactions, and enforces a pattern of thinking that may omit vital community dynamics (Olff et al., 2009).

In examples of this, mutualistic interactions between ants and aphids can affect the aphid-parasitoid network, causing a shift from generalist to specialist species (Sanders and van Veen, 2010). Environmental factors may also be important, such as in a host-parasite planktonic system where nutrient enrichment can affect host dynamics and lead to population cycles, altering chances of parasite persistence (Gerla et al., 2013), and parasites may influence the network structure and stability in turn, as can be observed when a decline in amphibians due to disease alters important ecosystem processes (Whiles et al., 2013). A final example demonstrates the benefits that considering multiple interaction types might offer, where it is suggested that ecologists could greatly increase efficiency by jointly managing pest control and pollination in agroecosystems (Fontaine et al., 2011). A good review of the necessity for considering multiple interaction types simultaneously, together with the importance of interaction type and intimacy on network architecture, may be found in Fontaine et al. (2011), and a suggested approach to tackling the inclusion of multiple interaction types in Kéfi et al. (2012).

The inclusion of multiple interaction types when considering ecological networks can have extensive consequences on factors such as the stability and structure of the network, as each interaction type has different effects on these measures (Allesina and Tang, 2012). The addition of parasitic species, without which ecological networks are incomplete, may alter the connectance and nestedness of a system, and therefore have consequences both for its robustness and its stability (Dunne et al., 2013; Freeland and Boulton, 1992; Lafferty et al., 2006b). Parasites can regulate host populations, and change many other theoretical properties of food webs that affect our understanding of them (Marcogliese and Cone, 1997). The introduction of parasites to a food web can also drive large changes in network structure (Britton, 2013), as witnessed in the fish species of a subarctic lake (Amundsen et al., 2013). In addition to this, the use of drugs on humans and animals (where coinfection with many parasites is ubiquitous) to eliminate certain parasite species could have an effect on the dynamics of other parasites in these hosts. This is a secondary aspect to drug use that concerns the effects of multiple interaction types, and it has only recently begun to be considered (Knowles et al., 2013).

Efforts have previously been made to include parasitic and other interaction types into the consideration of entire ecological networks, with mixed results. Joppa and Williams (2013) have used a niche model to include both antagonistic and mutualistic interactions in ecological networks, and although their accuracy decreases with network size, empirical properties across networks can often be closely approximated. It has also been suggested that the addition of parasites to food webs could increase their stability (Byers, 2009; Freeland and Boulton, 1992), although this may depend on the life-cycle of the parasite. If the life-cycle is simple, the parasite could be stabilising, while if it is trophically transmitted it could have a destabilising effect (Morand and Gonzalez, 1997). In addition, parasites could make food webs much less "robust", or resilient to secondary extinctions (Chen et al., 2011; Lafferty and Kuris, 2009). In general, it is know that the addition of parasites increases chain length and alters body-mass ratios, as well as introducing long loops of weak interactions as a result of complex life-cycle dynamics. Energy transfer from prey to predators may be reduced due to parasites, and population levels of common host species may be reduced due to the density dependence of parasites (Lafferty et al., 2008). Although all of the above will have an effect on the stability of a system, how exactly this occurs and the overall net effect is still often regarded as unclear.

Recently, Mougi and Kondoh (2012) addressed a similar problem regarding the mixing of antagonistic and mutualistic interaction types (see also Freeland and Boulton, 1992). They came to the conclusion that a mixture of interaction types increased the stability of a system. Although this conclusion may have been a result of the rescaling of interaction strengths (Suweis et al., 2013), the concept remains interesting. We propose here a model of an interaction network that includes parasitic interactions, and assess the effect that this has on the stability of the system. Unlike Mougi and Kondoh (2012), we do not investigate the local stability, but focus on the stability measured as the community persistence (see, for example, Brose et al., 2006 and James et al., 2012).

It is likely that linear stability analysis, as used in Mougi and Kondoh (2012), is not applicable to population dynamical equations of food webs, as real-world ecosystems are unlikely to be close to an equilibrium (Drossel and McKane, 2005). However, the community persistence that we consider here ensures that we can compare how many species are able to coexist in a

system, without being forced to extinction, which is a meaningful and comparable measure (see, for example, James et al., 2012). In addition to this, checking for local stability in the same manner as Mougi and Kondoh (2012) would be difficult for our system. The method that they use requires defining population growth rates such that the system is at equilibrium. In a predator-prey or mutualistic system, where growth rates may be negative if a species is not basal, this is straightforward, but this is not easily applied to a parasitic system. In such a system, the equivalent term, rate of recovery from infection, can only be positive. It may still be possible to select parameter values that allow for positive recovery rates using rejection sampling, but for larger systems this becomes increasingly difficult.

A third measure of stability, that we do not consider here either, is permanence (see Hofbauer and Sigmund, 1989; Jansen, 1987; Law and Blackford, 1992), a measure of the ability of species to increase when rare in a system, and hence to avoid extinction. Permanence is less limiting than local asymptotic stability, as it allows for systems with chaotic behaviour or limit cycles, similar to real ecosystems, to be classified as stable. However, it does have very stringent requirements for systems to be stable, considering all possible boundary points, which may not be the case in nature for a real "stable" system, and it also allows for very small population densities where in reality extinction might be observed (Law and Blackford, 1992). Most importantly, however, the inclusion of parasites in our model means that it is no longer straightforward to investigate permanence, as our system is not of the Lotka-Volterra form. We therefore do not consider permanence here. For further discussions on different types of stability, see Anderson et al. (1992); Chen and Cohen (2001) and Townsend et al. (2010), and for recent remarks on how this may relate to the diversity-stability debate, see Ives and Carpenter (2007) and McCann (2000)

We follow the method of Mougi and Kondoh (2012) in constructing a model with varying proportions of parasitic interactions, and we test this over mutualistic, antagonistic and mixed systems. We predict that the addition of parasites should decrease stability in our systems, as this is akin to the addition of top predators.

In general, we see that, as predicted, the overall levels of persistence in a system decrease across all interaction types with the addition of parasites. However, this does not tell the whole story. We discover that the effects of

parasitism on the persistence of free-living species depends on the interaction types that those species have, and while mutualists decrease in persistence on the addition of parasitic species, systems with mixed or predator-prey-only interactions experience relatively little change, with a potential increase in persistence. Parasite persistence is likewise affected by the interaction type of the network, with very little change in persistence even at high levels of parasitism in a predator-prey system, although other systems see significant decreases in persistence. This leads to a peak in parasite numbers able to exist in our system when the initial network is roughly two thirds parasitic.

7.1 The Model

Our model is based on the model of Mougi and Kondoh (2012), with the inclusion of microparasites, and is of an SIS form. We demonstrate this below for susceptibles X_i of host species i and infecteds I_{ij} of host species i infected with parasite species j, where $i \in \mathbb{N}_N$ and $j \in \mathbb{N}_P$. This results in a total population of host species i given by $H_i = X_i + \sum_{j=1}^{P} I_{ij}$. We have that

$$\frac{dH_i}{dt} = H_i\left(r_i - s_i H_i + \sum_{k=1}^{N} a_{ik} H_k\right) - \sum_{j=1}^{P} \gamma_{ij} I_{ij},$$

where

$$\frac{dX_i}{dt} = X_i\left(r_i - s_i H_i + \sum_{k=1}^{N} a_{ik} H_k - \sum_{j=1}^{P}\sum_{l=1}^{N} \beta_j^{il} I_{lj}\right) + \sum_{j=1}^{P} \rho_{ij} I_{ij},$$

$$\frac{dI_{ij}}{dt} = I_{ij}\left(r_i - s_i H_i + \sum_{k=1}^{N} a_{ik} H_k\right) + X_i \sum_{l=1}^{N} \beta_j^{il} I_{lj} - \gamma_{ij} I_{ij} - \rho_{ij} I_{ij}. \quad (7.1.1)$$

The model birth (r_i) and death (s_i) rates depend on the host species i, and there is an additional infection-induced mortality rate γ_{ij} of host species i due to parasite species j, as well as recovery rate ρ_{ij}. Infection with parasite species j occurs from hosts of species l to those of species i at rate β_j^{il}. Hosts of species i interact with host species k through coefficient a_{ik}, which may represent an antagonistic (predator-prey in this case) or mutualistic interaction. Note that both this term and the birth rate imply vertical transmission of parasites in our model, as the infected population is increased by birth or interaction with mutualistic or prey species.

7.2 Results

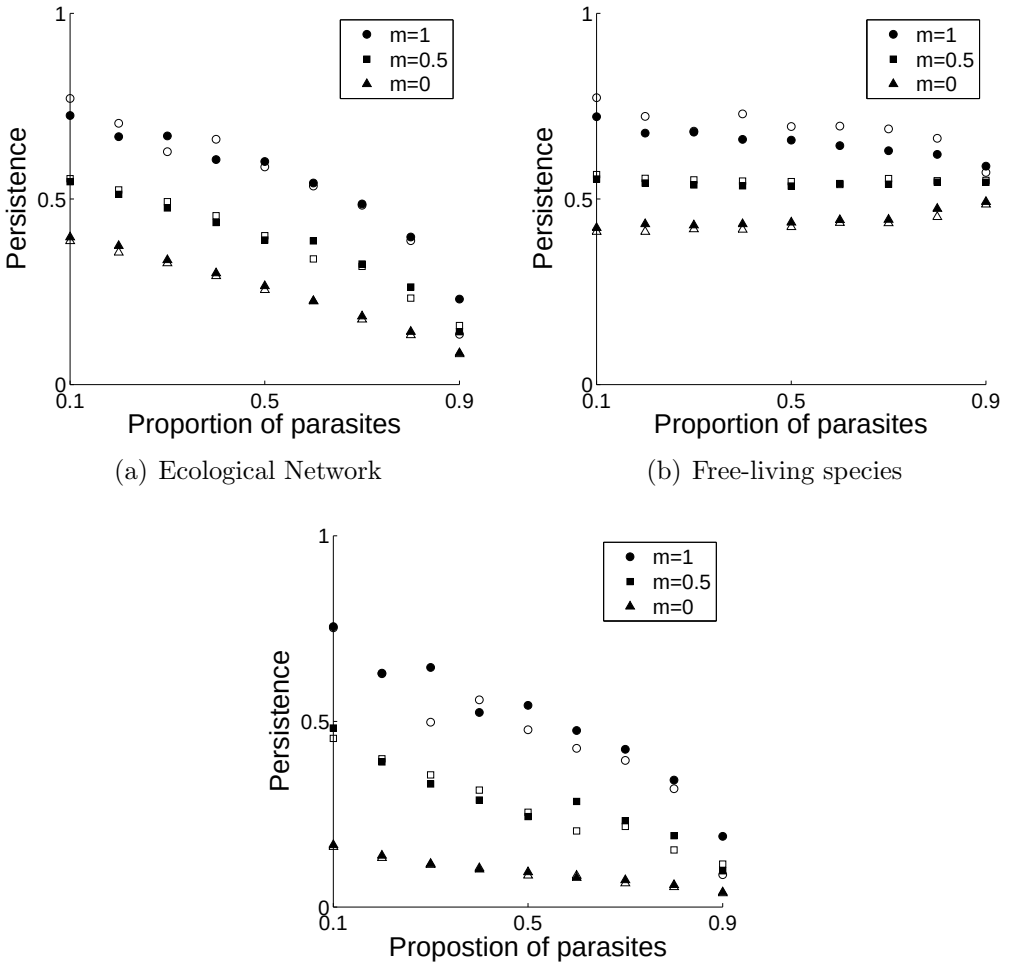
To test community persistence we choose each parameter and starting population size from a random uniform distribution between 0 and 1. Note that interaction strengths are random, and inversely proportional to the number of interactions, so a greater number of interactions for a species implies that these will be weaker. In addition to this, we structure the interactions according to the cascade model, so that there is a random trophic ordering on species, and species are only able to randomly consume those lower in the ordering than themselves (although there is no such ordering on mutualistic interactions, and we do not allow competitive or cannibalistic interactions). We then allow the population to equilibrate using Matlab (ode45 solver) according to system 7.1.1, and calculate the proportion of species that are able to coexist on average over 1000 systems. The results of this are presented in figure 7-1. In figure 7-2 we plot the same results as the total number of parasitic and free-living species that are able to exist for different proportions of parasitic interactions.

7.3 Discussion

From our results, it appears firstly that systems with higher levels of mutualism have higher levels of persistence, across all levels of parasite species richness. As the proportion of parasites in the system increases to very high levels, this effect is reduced, most likely because there are very few free-living species remaining in the system.

Although the overall persistence of the system, as well as the persistence of parasite species, decreases with an increasing proportion of parasitic species, there appears to be very little effect on the persistence of free-living species in general. We therefore conclude that an increase in proportion of parasites primarily reduces the persistence of parasite species, and that the majority of reduction in persistence of all species in the network is a consequence of this.

There is, however, still an effect that can be seen on the persistence of free-living species through the addition of parasite species, which depends on the level of mutualism. When the levels of mutualism are zero, then increasing parasite proportion is similar to increasing the number of top predators,

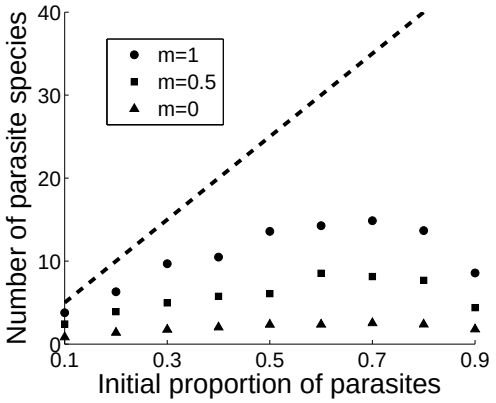


(a) Ecological Network

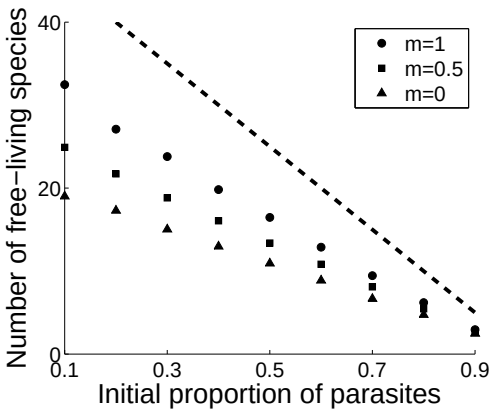
(b) Free-living species

(c) Parasite species

Figure 7-1: Average community persistence (proportion of species able to exist at ecological equilibrium) for ecological networks with different proportions of parasitic interactions, considering the persistence of (a) the entire ecological network, (b) free-living species only and (c) parasite species only. Different values of m represent different proportions of mutualism in the system. Here we consider for each proportion of parasitic interactions a total of 50 species over 1000 random systems, with parasites targeting 60% of species. Extinctions are considered to occur if population levels fall below 10^{-4}. The proportion of connected pairs is 0.4 for filled markers and 0.6 for bordered markers. We see that higher proportions of mutualism always lead to higher levels of persistence, and an increase in the proportion of parasites decreases levels of persistence for all species in all systems except free-living species in an antagonistic-only system.



(a)



(b)

Figure 7-2: Average number of (a) parasitic and (b) free-living species able to persist for different initial proportions of parasitic interactions in systems with 50 initial species (both parasitic and free-living). Different values of m represent different proportions of mutualism in the system. In each figure, the dashed line represents the initial number of species (parasitic or free-living respectively) that began in the system.

and the stability increases slightly. When mutualisms form roughly half of the interactions present in a system, then persistence is unaffected by parasites. However, when the system is mutualistic only, then the addition of parasites appears to decrease persistence of hosts. This is similar to the addition of predators to a mutualistic system, which we would expect to reduce persistence, as where there had previously been very little in the way of control in population growth for some species there could now be a limiting factor due to parasitic-induced mortality.

In addition to this, we note that the introduction of additional parasites to a predator-prey network does not come at as large a relative cost in terms of parasite persistence as for a mutualistic or mixed network. This may be seen by considering the slope of the line for an antagonistic-only system in figure 7-1(c). Similarly, we note from figure 7-2(a) that although parasite persistence decreases with proportion of parasites, the actual number of parasites able to persist increases only up to a point. It appears that there is a peak in the proportion of parasites in an initial system which allows for the highest number of parasites to exist, and the addition of further parasites at a cost to free-living species richness results in a reduction in parasite species persistence. From a parasite perspective, this suggests that an optimum exists at around two thirds of the population species richness, after which it is no longer feasible for additional parasite species to invade the system. This peak is more evident the more mutualists there are in a system, while in a predator-prey only system it appears that the number of parasites the system can support is relatively constant.

Although the introduction of parasite species simultaneously with the reduction in free-living species richness may confound the issue, it can be seen in figure 7-2(b) that, in general, the replacement of free-living species with parasites reduces the total number of free-living species able to persist in an ecosystem, although this is in part caused by the obvious initial decrease in species richness due to replacement with parasitic species. We observe that the slope of the best-fit line for final number of free-living species *versus* proportion of parasites is always less than the slope for the initial number, implying that the addition of parasitic species has less of an effect for higher proportions of parasitism in the system.

Similarly to the addition of mutualistic species to an ecological network (Mougi and Kondoh, 2012), we see that the addition of parasitic species does

significantly affect the stability of a network, as expected (Byers, 2009; Dunne et al., 2013; Freeland and Boulton, 1992; Lafferty et al., 2006b; Morand and Gonzalez, 1997). However, this is not a straightforward relationship. The interaction types in the network (mutualistic or antagonistic) alter the proportion of parasites that most favours free-living species persistence (see also Thébault and Fontaine, 2010). Some studies have investigated persistence for host-parasite interactions for small model (Wilson et al., 1996) and real-world (Tuda and Shimada, 2005) systems, but our results certainly could be difficult to test in an entire real-world ecological network. In addition, links between a system's stability and parasite species richness (Anderson and Sukhdeo, 2013a), as well as the effects of secondary extinctions (Colwell et al., 2012), confound this issue.

We had expected the introduction of parasites to destabilise our model, as parasites may be thought in some way to represent the addition of top predators. However, although parasites at first glance appear to destabilise the system, as in figure 7-1(a), on closer inspection we conclude that the effect of parasites depends on the proportion of mutualisms in the system. A system which is primarily mutualistic will be destabilised by parasites, while one which is primarily antagonistic will, in terms of free-living species, be slightly stabilised. The addition of parasites also appears to have less of an effect on the relative number of free-living species when levels of parasitism are already high. The effect of parasitic species on ecological networks, therefore, appears to depend on the nature of the network itself.

CHAPTER 8

Conclusions

In this thesis we have proposed theoretical ways in which the population and coevolutionary dynamics of host-parasite and mutualistic networks result in observed structural patterns. We see that an increase in the basic reproductive ratio could explain patterns of parasite species richness in downwardly asymmetric interactions, and we describe how a trade-off in resource use by species could lead to the patterns of significant nestedness and anti-nestedness observed in host-parasite networks, and of significant nestedness in mutualistic networks. We also determine the effect of parasitic interactions on the stability of ecological networks. This serves to highlight the importance of evolutionary and population dynamics in determining parasite species richness, and hence their importance as mechanistic explanations of parasite interactions in food webs. We see that structural features arise as a result of the dynamics of the system, where structural, dynamical and evolutionary aspects are all intrinsically linked. We also see that the pressure of evolution on species has a wide-spread effect on a system's dynamics, and indeed the structure and stability of entire ecosystems. In future work our approach might be expanded to include a greater number of species, interaction types and trade-offs (between such aspects as virulence and transmission).

In writing this thesis, we aimed to increase our understanding of the role and functioning of parasites in food webs, and in particular factors that lead to parasite species richness and the effects that this has. There are many

aspects that affect parasite species richness and diversity in hosts, depending on both the ecosystem and species involved. For example, the richness of tapeworms in elasmobranch fish can be explained by depth and latitude in shark species (Randhawa and Poulin, 2010), while Marcogliese (2002) has declared that parasite diversity is often due to the diversity of free-living organisms in a system and diversity in host diet. Parasite diversity may also be explained by the time since introduction of a host to the system, its body length, or its relatedness to native species (Paterson et al., 2012). Intriguingly, patterns of diversity and specificity of parasite species may also vary spatially, and specialists disappear along with species richness as one gets further from heartlands (Kennedy and Bush, 1994). However, the structure of interactions in a food web is widely acknowledged as being one of the most important determinants of parasite species richness and diversity (Marcogliese, 2001), although few studies have investigated this relationship (Anderson and Sukhdeo, 2011). Understanding the mechanisms behind this structure could therefore be vital in explaining ecological features of parasite diversity (Randhawa and Poulin, 2010).

In particular, dispersal and population structure are important determinants of local parasite adaption (Vogwill et al., 2009), implying that the structure of a food web will affect the evolution of parasites within it. Network measurements such as eigenvector centrality and trophic generality are consistent predictors of parasite diversity (Anderson and Sukhdeo, 2011), so parasites are often found in highly connected hosts, within modules of tightly interacting species. Host position in a food web also has a selective pressure on the evolution of parasitic transmission strategies, and hence patterns of diversity (Marcogliese and Cone, 1997; Poulin, 2010). In general, structural patterns of networks are important for their stability, and increasingly complex, non-random patterns of interaction strengths alone can increase the stability of networks (Neutel et al., 2007). Clearly, therefore, the structure of host-parasite networks is important in determining parasite species richness in food webs. Indeed, Anderson and Sukhdeo (2013b) note that, in a salt-marsh system, there is no linear relationship between the species richness of free-living species and of parasite species, which is more correlated with the structure and cohesiveness of the food web. In a reversal of this, parasitic interactions and parasite species richness may be important in determining food web structure, emphasizing the importance of considering all

of the interaction types involved in an ecological network (see Dunne et al., 2013; Kéfi et al., 2012; Leaper and Huxham, 2002; Dunne et al., 2013). The structure and dynamics of a food web are thus intrinsically linked, and must be considered in light of one another (Quince et al., 2005).

8.1 Outline

It is important for us to consider what drives parasitic interactions in food webs, and the effect that they have, as it is clear that ecological networks are incomplete without their inclusion (Byers, 2009; Lafferty et al., 2008, 2006b). We have chosen to focus on three particular aspects of the structure and stability of host-parasite interactions in food webs; nestedness, downward asymmetry and persistence. The first of these, nestedness, has been observed (along with its opposite, anti-nestedness) in host-parasite interactions of many different kinds (see Krasnov et al., 2005; Poulin, 2007a; Valtonen et al., 2001; Worthen and Rohde, 1996). Nestedness has been seen to increase with host range and to vary with host latitude (Krasnov et al., 2005). Recently it has been suggested that nestedness might be a by-product of processes such as coevolution or abundance, rather than because it is a more stable conformation type (Allesina, 2012), and the number of partners a species has may be of more importance than nestedness as a promoter of species coexistence (James et al., 2012). Pires and Guimarães (2013) claim that interaction intimacy in antagonistic networks causes patterns such as nestedness, with lower levels of intimacy leading to higher nestedness. It has also been suggested that nestedness is constrained and biased by the number of observations; rarely observed species are "specialists," while common species are often labelled "generalists," leading to observations of nestedness and asymmetry in interaction strengths occurring naturally as a result (Blüthgen et al., 2008). Despite this, there is still much evidence for nestedness (Graham et al., 2009; Joppa et al., 2010), and Vázquez et al. (2005) have constructed a brief model based on host abundance to account for this. Yet there may be many more possible explanations for the link between the specificity of a parasite and the parasite species-richness of the hosts that it infects, particularly as abundance cannot explain all observed patterns of nestedness and anti-nestedness that we see (Poulin, 2007b).

We have considered nestedness as a result of species trade-offs in inter-

actions, which have been used to recreate patterns of nestedness and anti-nestedness in host-parasite and mutualistic networks here. In a similar manner to our approach, Gilman et al. (2012) consider the coevolution of host and parasite traits, in which parasites must overcome all of a host's defensive traits in order to successfully infect it. The interaction probability depends on the trait values of both species interacting, either due to the difference in traits or to trait-matching. This favours host species, ensuring that they do not suffer unduly due to parasites, and gives an example of the integrated, host-parasite multi-species system approach that we feel we have achieved here, where natural selection acting on phenotypic traits determines patterns of interactions between species (see, for example, Beckerman et al., 2010).

The method that we have used is similar to the concept of optimal foraging, which may be important in stabilising population dynamics (Kondoh, 2003). Petchey et al. (2008) have also used optimal foraging theory to motivate consumer-resource interactions when looking at food webs, although they note that this performs better for herbivory and predation than for pathogens, parasites and parasitoids. In addition, Lozano (1991) describes how species use optimal foraging to protect themselves from parasites (i.e. individuals do not simply try to get the most nutrition possible from foraging, but try to get the most "fitness"; they try not to reduce their fitness due to parasitism). Indeed, many sources have demonstrated the manner in which host behaviour is altered by infection (Bethel and Holmes, 1977; Webber et al., 1987), as well as the trade-off that parasites must make when infecting different hosts (Ebert, 1998; Elena et al., 2009). From this we see that individual behaviour, and more specifically, who individuals interact with, is subject to natural selection from both a host and a parasite perspective. This determines individual interactions, and food web structure is the sum of these individual decisions. We model this phenomenon dynamically here, as Getz (2012) has done by looking at ordinary differential equations for the biomass of species. In fact, Getz (2012) has suggested that this could be a fruitful way to model parasites in food webs in the future, and we concur.

Many studies have looked at parasite and predator-prey species that can alter which species they attack or defend themselves against (Abrams and Kawecki, 1999; Matsuda et al., 1996; Osnas and Dobson, 2011), and many have investigated the trade-off between parasite virulence and transmission, amongst other factors (Frank, 1996; Frank and Schmid-Hempel, 2008; Osnas

and Dobson, 2011; Regoes et al., 2000). These can have far-reaching consequences, where the ecological dynamics can affect the presence of specialists or generalists, and their virulence or avirulence (Boldin and Kisdi, 2012; Regoes et al., 2000). Some studies have included certain aspects of the models that we discuss here, such as a study by Osnas and Dobson (2011), in which parasites experience a trade-off between virulence and transmission between different host species. It seems reasonable, therefore, that trade-offs in resource use are important to parasitic species. We note, however, that support for the theory of a trade-off in infectious potential is not ubiquitous or simple. For example, the adaption of tapeworms (*Schistocephalus solidus*) in a copepod (*Macrocyclops albidus*) and three-spined stickleback (*Gasterosteus aculeatus*) demonstrates very little trade-off in infective success (Hammerschmidt and Kurtz, 2005), where instead a trade-off in success over different parts of each host's immune system has been demonstrated.

Although this kind of trade-off is difficult to demonstrate biologically, mathematical models often include trade-offs at linked loci or due to antagonistic pleitropic allelic effects for resistance and infectivity (Osnas and Dobson, 2011). The manner in which trait values interact is also often unknown biologically, and models often assume them to be simply multiplicative or additive (see, for example, Osnas and Dobson, 2011; Regoes et al., 2000). In our model we assume that each immune system is completely unrelated and independent, and so host species may be regarded as entirely dissimilar objects, where trait values are multiplicative. Parasites vary in their ability to hide from or escape the host immune system, which varies in its response to different parasites (see again, for example, Osnas and Dobson, 2011). We then investigate the evolution of these trait values over time, and discover that this does indeed drive patterns of nestedness and anti-nestedness in different ecological networks, as predicted.

The coevolution of multiple hosts and parasites is of great importance, and can affect results significantly as hosts and parasites can have reciprocal effects on each other's phenotype and genotype (Ebert, 2008; Hood, 2003; Salvaudon et al., 2005; Webster et al., 2004) The coevolution of host and parasite interaction traits can therefore be used to describe and predict interactions in victim-exploiter relationships, and hence entire systems (Gilman et al., 2012, although see Kondoh et al., 2010). From this, the abundance and phylogenetics of the system can be seen, along with evolution, to determine

the nestedness of the system (Canard et al., 2012; Poulin, 2010), as we have modelled in Chapters 3, 4 and 5. Indeed, Fontaine et al. (2011) have emphasised the importance of ecology, evolution and coevolution in determining the architecture of sub-networks, such as those of hosts and parasites, and examples of a combination of evolutionary and ecological dynamics of ecosystems leading to structural features have recently begun to appear in the literature (see Fussmann et al., 2007; Ito and Ikegami, 2006; Quince et al., 2005). It appears from our results that, in agreement with previous work, the evolution of multiple trade-offs is key to understanding complex systems (Osnas and Dobson, 2011).

On the other hand, in Chapter 6 we are able to address a second structural feature of parasites in food webs by focusing on the population dynamics alone. This structural feature sees the interaction motif of downward asymmetry (where one predator species has many prey species) harbouring more trophically transmitted parasites than expected due to chance and diet breadth alone. These motifs are more likely to harbour parasites than symmetric interactions (with one predator and one prey species) because, although the latter guarantees transmission for a parasite with only one prey host species in its life-cycle, the strong links destabilise the system, leading to boom-and-bust dynamics (Rossiter and Sukhdeo, 2011). In addition, upwardly asymmetric interactions or weakly symmetric interactions (with many predator species and either few or many prey species respectively) see a dilution of parasites through predation, where they are often lost to non-host predators (Rossiter and Sukhdeo, 2011).

Rossiter and Sukhdeo (2011) accounted for increased parasite diversity due to the host having many prey species, and yet still found that hosts with many prey and few competitors collected more trophically transmitted parasites than expected due to chance alone. We explain this pattern mathematically, through an increase of the basic reproductive ratio for parasites in such hosts. The higher basic reproductive ratio of such parasites ensures that they are likely to be more successful than parasites found in other interaction motifs, and are more likely to be able to persist. Thus we are able to explain why more stable interactions enable parasite species persistence. Similarly to the concept of nestedness discussed above, we see that greater levels of success for parasite populations are the driving force behind structural features of parasites in food webs.

In Chapter 7 we consider the reverse; the effect that parasites have on the stability of food webs themselves. Here we examine the persistence of species, as a proxy for stability of the network. While the introduction of mutualists to a network has been thought to stabilise it, we see here that parasitic interactions have more complicated repercussions. The effect of parasites on persistence, it appears, is dependent on the level of mutualistic interactions already in the system. If this is high then persistence is reduced, while if this is low then persistence is increased. In terms of total species number, replacing free-living species with parasitic species reduces both the number of free-living species and the total number of species able to coexist. Part of this is caused by a peak in parasite species number persisting at an ecological equilibrium for a certain proportion of parasitic interactions in a network. Despite this, we are able to make a number of findings on the potentially destabilising effect of parasites, and conclude that their inclusion (or introduction) to a network may have far-reaching consequences, depending on the network structure and interactions already in place. In previous chapters we begin to answer the question "what drives parasite species richness, in both ecological and evolutionary terms?" In this final chapter, we answer the question that naturally follows on from this; "what are the consequences of this species richness?"

One of the major drawbacks in the mathematical modelling of large networks is the requirement of a large number of parameters, many of which it is often difficult to justify (Dobson et al., 2009, although see Goris et al., 2009). Hence, it is important to focus on robust, qualitative patterns, such as whether networks are more or less nested than expected due to chance, whether certain interaction types favour parasites, and the effects of interaction type on stability. Factors such as these could constrain web dynamics in unexpected ways (Montoya et al., 2006). In our work we have therefore focused on large-scale patterns that are evident across ecosystems.

8.2 Extensions

There are several ways in which our results could be extended. We have not included coinfection and recovery in our models of the coevolution of trait values. This removes the effects that parasites might have on one another while infection lasts, and no recovered class implies that we cannot assume

that species do not coinfect due to a short infection time, as there is no recovery from infection. In addition, our results in Chapters 4, 5 and 6 only includes small networks; we presume that patterns can scale up to larger systems, but computational constraints prevented us from testing this theory. We also have to bear in mind that, particularly for such larger systems, it is difficult to justify a choice of parameters, as discussed above. In the majority of our models, all species of hosts, parasites, plants and animals were assumed to be identical in every aspect, with the exception of trade-off strengths and initial trait values, in order to attempt to avoid the confounding effect that species with different life histories might introduce. However, in the real world species diversity is vast, and could lead to varied results. Although we have used few random, non-identical parameters (initial trait values and trade-off strengths only), the range of our results was large; imagine, then, the possible confusion that could result in a real-world network.

Additionally, the inclusion of multiple parasitic species to models of downwardly asymmetric interactions could help to elucidate whether the increase to the basic reproductive number applies to such cases, potentially supporting the claim that this leads to an increase in parasite species richness. We are able to see the importance of multiple-species coevolution in Chapters 3 to 5, implying that the inclusion of multiple predator and parasite species in Chapter 6 could be of similar significance. The addition of these species could alter the population dynamics considerably, and would allow for a full comparison with interaction motifs of many types. It has also proven challenging, in Chapter 7, to calculate the stability of ecological networks, an aspect that is confounded by the numerous ways in which stability itself may be defined, and how these relate to reality.

Perhaps the most interesting concept that we have discussed in this thesis is the evolution of interaction traits. Future work expanding this to large networks with multiple interaction types, and trade-offs in resources used for each, could represent a new way of thinking about ecological networks, and in particular the inclusion of parasitic species to these. A second exciting potential avenue of research is the possibility of empirically testing some of the theories suggested here, specifically those regarding nestedness. Trade-off strengths in bacteria and phage systems can be experimentally altered, and the effects that this has on the nestedness of these systems could be calculated to determine whether results are in line with our predictions. An interesting

extension to Chapter 6 would also be to model the system with multiple predators, in order to assess the effects of dilution, and, more importantly, multiple parasites. The second of these would confirm whether an increase in species richness, rather than simply species persistence that we discuss here, is indeed due to an increase in basic reproductive number. Finally, an assessment with a variety of other stability criteria in Chapter 7 could add to the results on persistence presented there.

8.3 Closing Remarks

Here, however, we are able to conclude that the dynamical modelling of food webs, taking into consideration individual-level mechanisms and coevolution, is key to the understanding of global structural properties. In order to understand how parasites fit into food webs, it is important to understand why they do so, and what drives them to display certain features of structure and interaction. We have also demonstrated that the consideration of resource use by individuals can be used to tie together many explanations of food web features. This is based on mechanistic principles, and describes forces that drive individuals and species on a daily basis, rather than professing over-arching "laws" such as the existence of forbidden links, or links between stability and species richness. Rather, we observe that such features as forbidden links are present as a result of the evolution of species over time, which results in individuals that are better able to compete and produce offspring, and species richness in stable interactions is a result of the increase in basic reproductive ratio for such species. In summary, species which produce a greater number of more viable offspring are statistically more likely to be successful, and it is by following the evolution of this phenomenon that we are able to describe and discuss the structure of the ecological networks that result. In this way, we have, as Allesina (2012) suggests, investigated large, weighted ecological networks by focusing on individual drivers and results.

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